Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

Posbus 643, Kaapstad P.O. Box 643, Cape Town

Kaapstad, 22 Maart 1958 Vol. 32 No. 12 Cape Town, 22 March 1958
Weekliks 2s. 6d. Weekly 2s. 6d.

PRIMARY PNEUMATURIA

WITH A REPORT OF A CASE DIAGNOSED RADIOLOGICALLY

R. GLYN THOMAS, M.B., B.CH. (RAND), D.M.R.D. (R.C.P. & S., ENG.)

and

A. SANDLER, M.B., B.CH. (RAND), M.R.C.P. (EDIN.)

Departments of Radiology and Medicine, University of the Witwatersrand and Johannesburg Hospital

Pneumaturia, namely the passage of gas from the bladder on voluntary micturition or during catheterization, is a rare phenomenon. The first mention of the condition was in 1671 in a publication entitled 'Curiosities of Nature', quoted by Taussig[1] as follows: 'A leading citizen of Gotha was afflicted by anal colic, with rumbling tension in the abdomen, pain about the navel, and, what is strange, wind was passed by the penis, as if through the usual and accustomed channel, sometimes with, and sometimes without urine.'

The aetiology may be secondary to introduction of air during instrumentation, or to a fistulous connection between the bladder and a gas-containing viscus such as bowel or vagina. Primary pneumaturia results from the formation of gas within the bladder as a result of urinary infection by a gas-forming organism. Rarely, pneumaturia may follow cystitis emphysematosa (see below), in which gas vesicles form within the bladder wall and subsequently rupture to liberate gas into the bladder cavity.

Fineman, Ferber and Roginsky[2] divide primary pneumaturia into 2 groups, viz. a group with glycosuria and a non-diabetic group.

Up to 1950, 18 cases of each group had been described according to these authors, who add reports of 4 additional diabetic cases with pneumaturia. Spring and Hymes[3] report a further diabetic case, contributing in addition an excellent review of the literature, and Foord and Nabarro[4] 2 additional cases associated with glycosuria, bringing the total of the reported instances of diabetic pneumaturia to 25.

Aetiology

Residual urine is almost invariably present owing either to the 'neurogenic bladder' of diabetic neuropathy or to bladder-neck obstruction in the non-glycosuric group. In Spring and Hymes' diabetic case[3], however, there was no evidence of incomplete bladder emptying.

Infection is superimposed on the vesical urinary stasis, and a gas is produced which is not readily soluble in urine. The infecting organism is usually *B. coli*, but *B. lactis aerogenes*, *B. aerobacter aerogenes*, *B. aerogenes capsulatus* and yeasts may be present. The urine is acid but becomes neutral or ammoniacal as fermentation proceeds.

Gas production may take place in the kidney and ureter as well as the bladder, but unless there is obstruction with urinary stasis in the kidneys no gas accumulation can occur since the urine passes rapidly into the bladder. Mueller[5] analysed the gas present in a diabetic patient with pneumaturia, and found it to contain carbon dioxide 9·1%, hydrogen 57·3%, nitrogen 33·5% and methane 0·79%. As the urine stands, more carbon dioxide is formed, and the proportion of this gas may reach 19%, while the hydrogen decreases with prolongation of fermentation. Hydrogen forms the bulk of the gas formed in diabetics, and carbon dioxide in the non-diabetic group. The gas is produced by fermentation in the glucose-rich urine with production of butyric and lactic acids. An appreciable quantity of alcohol may be produced on distillation of the urine.

There are several theories to explain the production of gas in non-diabetic urine. The gas may result from proteolytic break-down of a special type of albumen, or of ordinary albumen by a special strain of *B. coli*. Taussig[1] believed that the gas-producing ability of the coliform bacillus might be associated with a developmental phase of the organism. Mulsow and Gillies[6] suggest that acid urine from the kidneys may interact with bicarbonates present in the retained bladder urine with production of gas.

Diabetic cases may mistakenly be grouped as non-diabetic when the glucose is present in very small quantity or intermittently. Furthermore, glucose may be absent at the time of urine testing owing to its having been used up in the

fermentation process. A blood-sugar determination is therefore essential in patients exhibiting primary pneumaturia.

Ammoniacal fermentation or urine putrefaction does not produce pneumaturia because the gases are too readily soluble in urine.

The possible relationship of primary pneumaturia to cystitis emphysematosa deserves mention. This is an inflammatory condition, occurring more frequently in diabetics than non-diabetics, in which gas-filled vesicles appear in the walls of the urinary bladder as the result of *B. coli* infection. Pneumaturia is cited in case reports as a frequent symptom and, though Fineman, Ferber and Roginsky[2] are sceptical of a relationship, Boijsen and Lewis-Jonsson[7] believe that free gas in the bladder follows rupture of the vesicles in the late stages of emphysematous cystitis.

Diagnosis

The clinical diagnosis is suggested by the finding of a mid-line supra-pubic tympanitic 'mass' which may reach the level of the umbilicus. The patient complains of the passage of gas during micturition, or the condition may be found on catheterization or abdominal radiography without the patient's being aware of it.

Reference in the literature to the radiological diagnosis has been made only 7 times so far. The radiological features to be noted are:

1. A round or pyriform translucency in the supra-pubic mid-line area, distinguishable from the sigmoid and rectal gas-shadows or not resembling bowel gas-shadows (Fig. 1).

2. A pelvic fluid level with gas cap above it when a horizontal X-ray beam is used with the patient in the erect position.

3. On excretory urography, the opaque medium pools in the dependent portion of the bladder in the supine position, giving the appearance of a poorly defined blob of opacity, surrounded by a broad halo of translucent gas. In the erect posture there is a fluid level at the upper limit of urographic medium, with gas cap completing the bladder outline.

4. Any translucencies in the region of the urinary bladder in the plain abdominal film should raise suspicion of gas in the bladder, particularly if bowel shadows can be separately distinguished. Often it is impossible to distinguish between bowel and bladder gas, and catheterization must be resorted to as the final arbiter.

CASE REPORT

Mr. C.J.M., a deaf-mute aged 53, suffering from primary testicular atrophy and eunuchoid gigantism for many years, had diabetes mellitus, present for the past 10 years. The diabetes was always difficult to control with insulin, and he had been admitted to hospital on numerous occasions either in hypoglycaemic or hyperglycaemic coma.

The patient was in hospital convalescing from an episode of hypoglycaemic coma, when he developed burning micturition, and profuse haematuria which lasted one week. When the haematuria ceased, the urine contained traces of albumen and varying amounts of glucose. Cultivation of mid-stream specimens of urine and subsequent catheter specimens yielded abundant growths of coliform bacilli sensitive only to neomycin. Straight X-ray of the abdomen revealed the gas shadow in the bladder seen in Fig. 1. In view of the X-ray picture, the bladder was catheterized under water, when first urine and then gas were obtained. Insufficient gas was obtained for laboratory analysis. On rectal examination, the prostate was found to be small. There was no evidence of a fistulous connection between the bowel and the bladder.

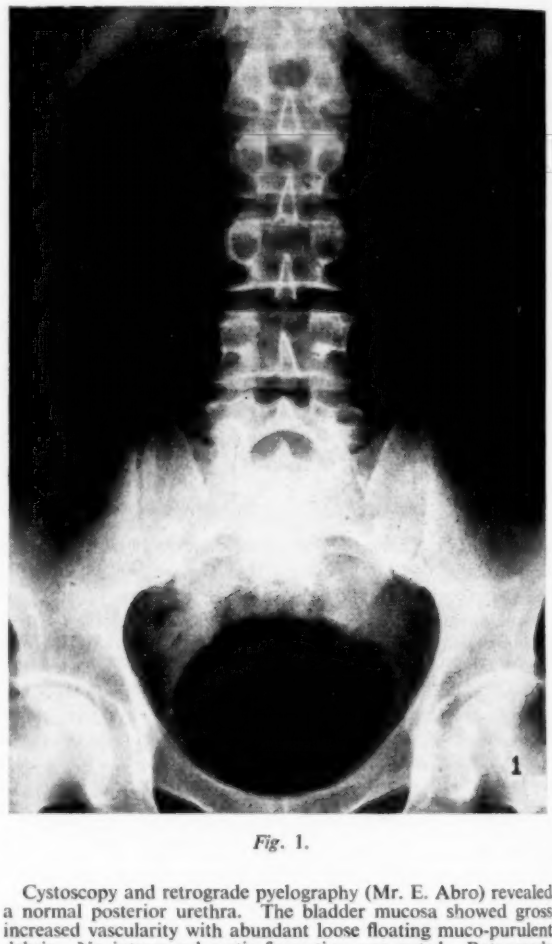

Fig. 1.

Cystoscopy and retrograde pyelography (Mr. E. Abro) revealed a normal posterior urethra. The bladder mucosa showed gross increased vascularity with abundant loose floating muco-purulent debris. No intramural cystic formation was noted. Retrograde pyelography revealed normal calyceal systems with some pyelectasis but no renal papillary necrosis. There appeared to be narrowing of the pelvi-ureteric junction, but the emptying was normal. Ureteric dilatation was present, consistent with bladder infection.

Neomycin was instilled daily into the bladder. The pneumaturia disappeared, but the coliform infection persisted. The daily lente insulin requirement as calculated by urinary sugars was 40 units before the onset of the haematuria and pneumaturia and 15 units during the episode, and gradually increased to 50 units when the pneumaturia disappeared. This is contrary to what is expected in a diabetic who develops secondary infection, at least insofar as control by urine testing is concerned. The blood sugars before, during and after the pneumaturia episode in this case were not studied in enough detail to permit of correlating the blood-sugar levels, the presence or absence of gas in the urine, and the insulin requirements and dosage.

SUMMARY

Primary pneumaturia is a rare phenomenon. It occurs slightly more commonly in diabetics than in non-diabetics, and is usually due to coliform bacillus infection. The clinical and radiological features of a case are described. It is important to recognize the condition because, apart from the obvious

implication of infection of the urinary tract, urine testing may give fallacious results because of the using up of the glucose by the infecting organisms. Clinical control of the diabetes can only be adequately effected by frequent blood-sugar estimations.

Thanks are due to Dr. Josse Kaye, Chief Radiologist, Johannesburg General Hospital, and Prof. G. A. Elliott, Professor of Medicine and Senior Physician, Johannesburg General Hospital, for their permission to publish this case.

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A NOTE ON THE INCIDENCE OF BILHARZIA IN DURBAN BANTU SCHOOL CHILDREN

L. FREEDMAN, B.Sc., PH.D.

Research Officer, Amoebiasis Research Unit, Durban*

and

R. ELSDON-DEW, M.D., F.R.S.S.AF.

Director, Amoebiasis Research Unit, and Senior Lecturer in Parasitology, University of Natal, Durban*

A single specimen of urine from each child in a Bantu co-educational school in Durban was examined for *Schistosoma haematobium*. The results are given in the following table:

Age	Male			Female			Total		
	No.	+	%	*No.*	+	%	*No.*	+	%
0- 9	126	28	22·2	19	5	26·3	145	33	22·8
10-14	264	149	56·4	248	49	19·8	512	198	38·7
15+	127	65	51·2	89	18	20·2	216	83	38·4
Total	517	242	46·8	356	72	20·2	873	314	36·0

This table shows not only that ova are found more frequently in males, but that the difference between the sexes varies with the age-groups. Younger females have an incidence similar to that of males in their own age-group, but with increasing age the females show no obvious increase in incidence whereas the males show more than double the number of infections.

The uncleanly habits of the subjects were evidenced by the finding of such parasites as *Ascaris*, *Taenia*, *Trichocephalus* and *Enterobius* in the specimens.

The examination of a single random urine specimen cannot give the true incidence of *S. haematobium*, which is probably at least 50% higher. Nevertheless, the figures indicate the prevalence of the condition in the Bantu even under urbanized conditions.

We have to thank Dr. A. Stephen and the staff of the City Health Department, Durban, for their co-operation.

* The Amoebiasis Research Unit is sponsored by the following bodies. The South African Council for Scientific and Industrial Research, the Natal Provincial Administration, the University of Natal, and the United States Public Health Service (Grant E-1592).

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VAN DIE REDAKSIE

DIE JONGSTE TOETSE VIR LEWERFUNKSIE

Hoewel die biochemiese toetse wat tans gebruik word om lewerfunksie te toets, ondersteun waar nodig deur naaldbiopsie, dit gewoonlik moontlik maak om die oorsaak van geelsug of lewervergroting vas te stel, kom ons nogtans dikwels diagnostiese struikelblokke teë by die onderskei tussen obstruksies *buite* en *binne* die lewer. Verskeie nuwe toetse is in die afgelope tyd gebruik hopende dat hulle by sulke probleemgevalle nuttig sal wees.

Die berekening van die glutamienoksaalasynsuur-transaminase in die serum het vandag 'n vasgestelde waarde by die diagnose van lewersiekte sowel as van hartspierverstopping.[1] Dit styg geweldig na ernstige lewerbesering, maar by eenvoudige obstruksiegeelsug is dit net effens hoër as die normale; by sirrose varieer dit geweldig. Die korrelasie tussen hierdie toets en ander ‚lewerfunksie-toetse' is maar swak; 'n verhoogde transaminase-gehalte dui blykbaar op vernietiging van lewerselle.

Die reaksie op ACTH is nog 'n nuttige toets: by pasiënte met obstruksie-geelsug veroorsaak deur virus-lewerontsteking (‚galbuis-lewerontsteking') daal die bilirubien en alkaliese fosfatase in die serum;[2] geen soortgelyke reaksie is dusver gerapporteer by gevalle van verstopping van die galweë binne die lewer weens ander oorsake, soos bv. chlorpromazine nie. Indien die serum-bilirubien daal wanneer steroïede toegedien word aan 'n pasiënt met 'n verstopping buite die lewer, styg dit weer as die hormoontoediening gestaak word.[3]

Volgens Rachmilewitz *et al.*[4] stort die beskadigde lewerselle opgegaarde vitamien B_{12} in die bloedsomloop uit; na bewering word hoë gehaltes B_{12} in die serum teëgekom by akute virusontsteking van die lewer, maar *nie* by buitelewerse galwegverstopping met gepaardgaande geelsug nie.

Die kleurstof Rose Bengal verbind met die serumproteïne ná binneaarse inspuiting en word dan deur die lewer uit hierdie proteïne geëkstraheer. Dit kan met ^{131}I gemerk word; dit word gemeen dat die snelheid waarteen die radioaktiewe verbinding deur die lewer opgeneem word (aangetoon deur eksterne kontrole) 'n nuttige toets is van lewerfunksie.[5] Hierdie toets is na bewering ook nuttig om buite- van binnelewerse verstopping te onderskei.[6]

Nog 'n nuwe toets is die berekening van die konsentrasie van sekere galsuurfraksies in die serum.[7] Normaalweg is die peil van trihidroksie en dihidroksie galsure in die serum 1·4 en 0·4 mg. per 100 ml. respektiewelik. By ernstige lewerbeskadiging swaai die verhouding om; as dit so aanhou is 'n swak prognose te verwagte. By verstopping buite sowel as binne die lewer styg die konsentrasie van beide fraksies en bly die verhouding konstant. Hoewel hierdie

EDITORIAL

NEWER TESTS OF HEPATIC FUNCTION

While the currently-used biochemical tests of liver function, reinforced where indicated by needle biopsy, usually enable the cause of jaundice or hepatomegaly to be discovered, diagnostic difficulties often exist in the differentiation of extrahepatic and intrahepatic obstruction. Several tests have recently been introduced in the hope that they will prove useful in these problem cases.

Estimation of the serum glutamic oxalacetic transaminase is now of established value in the diagnosis of liver disease as well as of myocardial infarction.[1] It is greatly elevated after acute liver injury but only slightly so in pure obstructive jaundice; in cirrhosis the levels vary widely. Correlation with other 'liver function tests' is poor; an elevated transaminase value appears to indicate liver-cell destruction.

Another useful test is the response to ACTH: in patients with obstructive jaundice due to viral hepatitis ('cholangiolitic hepatitis') the serum levels of bilirubin and alkaline phosphatase fall;[2] a similar response has not yet been reported in cases of intrahepatic cholestasis due to other causes, e.g. chlorpromazine. Should the serum bilirubin fall when steroids are given to a patient with extrahepatic obstruction it rises again once the hormones are stopped.[3]

According to Rachmilewitz *et al.*, damaged liver cells release stored vitamin B12 into the circulation; high serum-B12 values are said to be found in acute viral hepatitis, but not in extrahepatic biliary obstruction with jaundice.

The dye Rose Bengal becomes bound to the serum proteins after intravenous injection and is then extracted from them by the liver. It can be labelled with ^{131}I; the rapidity with which the radio-active compound is taken up by the liver (as shown by external monitoring) is thought to be a useful test of liver function.[5] This test is also said to be helpful in distinguishing extrahepatic from intrahepatic obstruction.[6]

Another new test is the estimation of the concentration of certain bile-acid fractions in the serum.[7] Normally the serum levels of trihydroxy and dihydroxy bile acids are 1·4 and 0·4 mg. per 100 ml. respectively. With severe liver injury the ratio becomes reversed; should this persist, the prognosis is likely to be poor. In both extrahepatic and intrahepatic obstruction the concentration of both fractions is increased

toets dus blykbaar nuttig is om die erns van die lewerbeskadiging en die hoop op herstel te bereken, help dit nie baie om die plek waar die galwegverstopping plaasvind, te bepaal nie.

Sommige van hierdie toetse is ook nuttig by gevalle waar uitsaaiingsgewasse van die lewer vermoed word. Dit is reeds al 'n hele rukkie bekend dat sekondêre neerslae in die lewer dit moontlik maak vir ensieme soos alkalasie fosfatase om in die bloedsomloop te kom. Na bewering is 'n styging in die serum-transaminase 'n sensitiewe maatstaf van die teenwoordigheid van neoplastiese neerslae in die lewer;[1] maar hoë transaminasegehaltes kom by verskeie siektes voor. Dit is ook bewys dat die vitamien B_{12} in die serum styg wanneer daar uitsaaiingsgewasse in die lewer is (dit kom nie voor by kwaadaardigheid sonder sekondêre lewergewasse nie).[8]

Dit lyk dus of hierdie toetse van praktiese waarde kan wees, en hulle vestig bowendien die aandag op aspekte van lewerfunksie wat nog nie goed verstaan word nie. Dit is heel moontlik dat hul gebruik by moeilike gevalle van lewerkwale baie lonend kan wees.

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and the ratio is unchanged. Thus, while this test appears to be of value in assessing the severity of liver damage and the likelihood of recovery, it is less useful in indicating the site of biliary obstruction.

In addition, some of these tests are helpful in cases where metastatic tumours of the liver are suspected. It has for some time been known that secondary deposits in the liver may enable enzymes, e.g. alkaline phosphatase, to enter the circulation. Elevation of the serum transaminase is said to be a sensitive index of the presence of neoplastic deposits in the liver;[1] but high transaminase values are found in a variety of disorders. It has also been shown that the serum vitamin-B12 level is raised when there are metastases in the liver (this is not found in malignancy without liver secondaries).[8]

Not only do these tests seem likely to be of practical use, but they also focus attention on ill-understood aspects of liver function. Their application to difficult cases of hepatic disease may well be rewarding.

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DIPHTHERIA PROPHYLACTICS*

THEIR PREPARATION AND ASSAY IN THE LABORATORY

J. H. MASON

South African Institute for Medical Research, Johannesburg

The real fight against diphtheria began in 1884 when Loeffler[1] isolated *Corynebacterium diphtheriae* in artificial culture. This made possible the preparation of toxin by Roux and Yersin[2] in 1888, and of antitoxin in small animals by von Behring and Kitasato[3] in 1890 and in horses by Roux and Martin[4] in 1894. Ehrlich[5] in 1897 introduced methods of standardizing toxin and antitoxin by *in vitro* methods (L+ and Lo), coined the name 'toxoid' and studied this non-toxic derivative of toxin in detail. Ramon[6] in 1922 showed that toxin and antitoxin may be titrated accurately and quickly by an *in vitro* flocculation test (Lf). Schick[7] introduced the test named after him in 1913.

In 1913, Von Behring[8] used toxin-antitoxin mixtures (TAM) for the immunization of man and in 1914 Park and Zingher[9] in New York City carried out an extensive immunization programme with this prophylactic.

Glenny and Südmersen[10] and Glenny and Hopkins[11] (1923) in Britain and Ramon[12] (1923) in France prepared formol-toxoid (FT) and in 1926 Glenny and his colleagues[13] introduced the first 'particulate' prophylactic, alum-precipitated toxoid (APT). Holt[14] in 1946 prepared another particulate antigen, PTAP—purified toxoid adsorbed on aluminium phosphate ($AlPO_4$) although, before this time, less thoroughly purified FT adsorbed on aluminium hydroxide had been used, particularly in Denmark.

Hartley[15] (1925) introduced toxin-antitoxin floccules and Glenny and Pope[16] (1927) prepared toxoid-antitoxin floccules (TAF). In 1951 Mason[17] introduced ADF—TAF dissolved in alkali and adsorbed on $AlPO_4$.

The first antitoxins in general use were the 'natural' sera of hyperimmunized horses. Later, some degree of purification and concentration was obtained by salting out the antitoxin-containing globulins with ammonium or sodium sulphate, and in recent years a marked purification and concentration has been achieved by 'refining' the antitoxic serum with pepsin (Parfentjev,[18] 1936; Pope,[19] 1938-1939; Hansen,[20] 1941).

A bibliography of all the articles leading up to modern methods of preparing prophylactics would, alone, run to many pages. Those given have an important bearing on the discussion of methods that follows.

PREPARATION OF TOXIN

Strain of C. diphtheriae. There can be few production laboratories that do not use the classical Park-Williams 8

* Presented at the South African Medical Congress, Durban, September 1957.

(PW8) strain, or a variant of it, for toxin production, because a 'wild' highly toxigenic strain is seldom encountered. In a recent investigation of 193 such strains isolated from patients in the Fever Hospital, Johannesburg, none was found to approach PW8 as a toxin producer and an attempt to 'coach' a few likely ones to produce potent toxin was unsuccessful. In these laboratories, the 'Toronto' variant of PW8 is used.

Culture Medium. The composition varies from laboratory to laboratory but, as a rule, it is an enzymatic digest of ox flesh or casein or an acid hydrolysate of casein, supplemented with amino acids, a source of carbon such as sodium acetate or lactate, salts, and maltose. The iron content is of prime importance for good toxin production and unless the medium is of a purely synthetic origin, it is nearly always necessary to carry out deferration so that the medium contains less than 0·2 μg. iron/ml. The medium used in these laboratories is a slight modification of that devised by Pope and Linggood[21] (1939)—a pancreatic digest of ox flesh, to which is added sodium acetate, sodium lactate, yeast extract and maltose.

Culture Vessels. C. diphtheriae is a strict aerobe and does not produce potent toxin unless the oxygen supply is adequate. For this reason, medium is placed in flat-bottomed flasks to form a layer not more than 2 cm. deep.

Incubation. 'Still' cultures are incubated at 35°C (not 37°C) for 6 days. A thin, translucent pellicle covers the surface of the medium in 24-36 hours, thickens considerably in 48 hours, and later forms 'curtains'. By the 6th day, a copious precipitate of deposited bacteria is present and the medium has a sherry colour due to the formation of porphyrins. Linggood *et al.*[22] (1954) have introduced an ingenious modification whereby the potency of toxin may be increased 3-4 times and the period of incubation reduced to 2 days. 'Double-strength' broth containing 4-8 times the usual amount of maltose is dispensed, not in shallow layers, but in 14 litre quantities in containers containing a propeller and an air inlet. Sterile air is introduced, is sucked down the vortex and is forced into the medium by the propeller in the form of minute bubbles. A quick, luxuriant growth, with high toxin production is the result. The method has been adopted in these laboratories.

Harvesting and Sterilization of Toxin. The cultures in the flasks are bulked, clarified through cloth coated with infusorial earth, and sterilized by candle or pad filtration.

THE TITRATION OF TOXIN AND TOXOID

Minimal Lethal Dose of a Toxin (*m.l.d.*)

This is the smallest amount that, injected subcutaneously, will kill a 250 g. guinea-pig within 5 days. This test measures the *toxicity* of a toxic filtrate but not its power to unite with antitoxin, or its antigenicity, i.e. its power to produce immunity.

Under the heading, 'Formol-toxoid', the preparation of 'artificial' toxoid with formalin is discussed, but what may be called 'natural toxoid', i.e. a non-toxic derivative of toxin, is present even in freshly prepared toxic filtrates and it increases in amount as the material becomes older. Thus, the m.l.d. will increase with the passage of time.

A toxin with an m.l.d. of $x/100$ ml. will most probably be, either as toxin or toxoid, a better antigen than one with an m.l.d. of x ml., but it does not follow that one with an m.l.d. of $x/2$ ml. will be twice as good an antigen as one with an m.l.d. of x ml.

The toxic filtrate with an m.l.d. of x ml. *may* contain much more natural toxoid than that with an m.l.d. of $x/2$ ml.; if so it will combine with more antitoxin and thus, other things being equal, has a better chance of being a better antigen (see under 'L+', 'Lr', 'Lf' and 'Laboratory Assay of Prophylactics').

In the preparation of prophylactics, there is little need to find the m.l.d. of a toxin because it furnishes very little useful information.

Minimal (Skin) Reacting Dose of a Toxin (*m.r.d.*)

This is the smallest amount of toxin that will cause a reaction within 48 hours when injected intracutaneously into a guinea-pig or rabbit. It is a much smaller amount than the m.l.d. and will, therefore, detect less antitoxin but, as for the m.l.d., there is no need, in routine toxin production, to carry out the test (see 'Lr' and 'Schick toxin').

L+ and Lo (L=limit; +=death sign; o=null sign)

The symbol L+ represents the smallest amount of toxin which, mixed with 1 unit of antitoxin and injected subcutaneously, will kill a 250 g. guinea-pig within 5 days.

The Lo is the largest amount of toxin which, mixed with 1 unit of antitoxin and injected subcutaneously, will not cause a local oedema in a guinea-pig.

Until the introduction of the flocculation test, the estimation of the L+ was the only means of finding the antitoxin-binding power of a toxic filtrate, because it also measures the toxoid content as toxoid is bound by, or unites with, antitoxin in the same way as toxin. The lower the L+ of a toxin, the higher is its antitoxin-binding value; for example, if the L+ of toxin A is 0·01 ml. and of toxin B 0·04 ml., then 1·0 ml. of A is bound by or is equivalent to 100 units of antitoxin whereas 1·0 ml. of B is equivalent to 25 units, a 4-times difference in favour of A. Experience has shown that, other things being equal, the higher the antitoxin-binding power of a toxin, the better will be its antigenicity and that of the FT prepared from it; but this value is obtained nowadays more quickly and accurately by the flocculation test.

Lr of a Toxin (r=reaction)

This is the smallest amount of toxin which, mixed with 1 unit of antitoxin and injected intracutaneously into a guinea-pig or rabbit, will cause a reaction within 48 hours. This test has the advantage over the L+ that 10-16 titrations may be carried out in one guinea-pig and 40-60 in one rabbit. But, as with the L+, there is little need, as a routine method, to find the Lr of a toxin, because the flocculation test has replaced it. However, the Lr/1000 dose of Schick toxin must be found, and the intracutaneous test is used, as a routine, to titrate antitoxin for issue (see below) and to assay sera the antitoxin content of which is too small to be found by flocculation.

Lf of a Toxin or Toxoid (f=flocculation)

This is the amount of toxin or toxoid that is equivalent to 1 unit of antitoxin as determined by the flocculation test. By experience one knows that a routinely produced toxin will have an Lf of between 60 and 100, i.e., 1 ml. will be equivalent to between 60 and 100 units of antitoxin. A unit

of antitoxin has been laid down by an international body, and standard antitoxin containing a stated number of units per ml. is issued to laboratories on request, every 6 months. With such a standard or with a laboratory standard checked against it, the Lf, L+ or Lr of a toxin may be established.

The test is carried out in the following way: Into a series of small tubes, 50, 55, 60 . . . 110 units of antitoxin and 1 ml. of toxin or toxoid under test are pipetted. The contents are mixed and the tubes placed in a water bath at 42°C. They are inspected frequently and carefully. After a period varying, according to the antigen and/or the antitoxin, from a few seconds to an hour or two, an opalescence and later floccules will be noted in one tube, the *initial* tube; still later, flocculation will take place in the tubes to the right and left of this tube. If the initial tube contains 80 units of antitoxin, the Lf of the toxin is 80, i.e. 1 ml. of toxin is neutralized by, or is equivalent to, 80 units of antitoxin. In the supernatant of this flocculated mixture, no free toxin or antitoxin (or only a trace of one or the other) will be found. This test is rapid and accurate and assays *toxoid* as well as toxin. Further, if toxin or toxoid of known Lf value is used, antitoxin may be evaluated equally quickly. However, for reasons that will be discussed later, the flocculation test must not be used as a measure of the antigenicity of a prophylactic or of the curative value of an antitoxin.

PREPARATION OF PROPHYLACTICS

Formol-toxoid (FT)

This is the basis of all prophylactics in use today. To toxin, enough formalin (40% formaldehyde) is added to combine with the free amino acids plus an additional amount found by experience; in these laboratories, the total amount added varies between 0·7 ml. and 0·9 ml. per 100 ml. toxin. The pH is adjusted to 7·2, phosphate buffer to maintain this pH is added, and the bottles are incubated at 37°C for 30 days. On 3 occasions at weekly intervals the pH is checked and brought to 7·2 if necessary. A high pH causes considerable destruction of toxin as opposed to conversion to toxoid. At the end of the 'toxoiding' process, a sample is tested got non-toxicity (5 ml. injected subcutaneously must cause no more than a transitory swelling in a guinea-pig) and for Lf value. As a rule, this falls slightly owing to destruction of antigen.

Until the introduction of APT and later of PTAP, FT was the only prophylactic used; it is still used, usually in a purified form, frequently combined with *Haemophilus pertussis*. It produces a satisfactory but not a high-grade immunity after 3 injections of 50 Lf at monthly intervals. Like most diphtheria prophylactics it is well tolerated by babies but is liable to cause reactions in some older children and adolescents.

Alum-precipitated Toxoid (*APT*)

Some degree of decolorization (removal of porphyrins) and purification of FT is usually carried out, e.g. by treatment with charcoal or Decolorite (an ion-exchange resin). The optimum amount of potash-alum to be added is ascertained, i.e. that amount that will precipitate the largest amount of antigen with the smallest nitrogen content. This amount, usually between 1% and 2%, is added to the bulk. The precipitate that forms is washed 4 times in saline and finally suspended in saline so that 0·5 ml. of the prophylactic contains 25 Lf found by flocculation after dissolving the precipitate in 2% sodium-citrate solution.

APT deservedly enjoyed a world-wide use and is only now being superseded by PTAP. It produces a solid basic immunity after 2 injections, each of 25 Lf, separated by an interval of 4-8 weeks. Its liability to cause reactions is similar to that of FT.

Purified Toxoid Adsorbed on Aluminimum Phosphate (*PTAP*)

Methods of purification vary from laboratory to laboratory and include removal of pigment on magnesia gel, charcoal or ion-exchange resin; concentration by ultra-filtration or fractional precipitation by ammonium sulphate followed by dialysis; further fractional precipitation by ammonium sulphate; and adsorption on, and elution from, cadmium chloride followed by dialysis. It is relatively easy to increase the purity of a crude toxoid of 150-200 Lf/mg. protein nitrogen (PN) to 1800-2000 Lf/mg. PN, but much more difficult and somewhat wasteful to increase this to 2500-3000 Lf/mg. PN.

In these laboratories, most of the colour and a considerable amount of nitrogenous impurity is removed on charcoal and on Decolorite; 40 g. of ammonium sulphate—$(NH_4)_2SO_4$—is added to each 100 ml. of decolorized toxoid, the precipitate is collected and washed with 40% $(NH_4)_2SO_4$ and then dialysed in a Cellophane bag against very cold running water for 5 days. The contents of the bag are clarified and the toxoid is precipitated with 0·75% cadmium chloride. After being washed in 0·5% cadmium chloride the precipitate is dissolved in 5% sodium phosphate (Na_2HPO_4). The solution is dialysed for 4 days against cold running water, clarified, made isotonic with NaCl, preserved with 0·01% Merthiolate, and finally sterilized by candle filtration. The purity varies between 1,800 and 2,000 Lf/mg. PN.

Purified toxoid is, Lf for Lf, no better and probably worse than crude FT as an antigen, but when adsorbed on $AlPO_4$ it is much superior. It is added as a concentrate (4,000-6,000 Lf/ml.) to an $AlPO_4$ suspension at pH 5·7 so that the PTAP for issue will contain 25 Lf and 2·5 mg. $AlPO_4$ per 0·5 ml. A period of 3 and preferably of 6 months' storage at about 20°C is necessary for a firm adsorption of the toxoid to the $AlPO_4$ to take place.

PTAP is a very good antigen, producing a solid basic immunity after 2 injections of 25 Lf at 4-8 weeks' interval. It causes fewer reactions than FT or APT.

Dissolved Toxoid-antitoxin Floccules (*DF*) *and Adsorbed Dissolved Floccules* (*ADF*)

On *a priori* grounds it could be assumed that the diphtheria toxoid moiety of floccules would be purer than a toxoid purified by physico-chemical means. If toxoid alone flocculated from a crude FT, it would be absolutely pure but, unfortunately, other constituents 'come down' in the floccules. But by gel diffusion tests, it has been shown that DF is purer than purified prophylactics regularly issued. By a gel diffusion technique, Pope *et al.*[23] (1951) demonstrated 14 antigens in a toxoid containing 2,170 Lf/mg. N whereas, by a similar method, no more than 4 have been found in DF.

DF is prepared by flocculating FT with refined antitoxin at the 'initial tube' level. The floccules are spun out and washed 4 times with saline. They are then dissolved in N/20 NaOH and the alkali allowed to act for 1 hour at room temperature. This treatment destroys all but a minute

trace of the antitoxin. The alkali is neutralized with HCl, and NaCl to isotonicity and 0·01% Merthiolate are added; the concentrated DF solution is sterilized by candle filtration. Thereafter, the adsorption on to $AlPO_4$ to produce ADF is the same as that noted under 'PTAP'.

The immunity produced by 25 Lf of ADF followed in 6-8 weeks' time by another 25 Lf of ADF or, preferably, of DF is of the same order as that obtained with PTAP. Possibly it produces fewer reactions than PTAP in older children and adolescents.

Combined Prophylactics

PTAP may be mixed with a suspension of *H. pertussis* or with *H. pertussis* and purified tetanus toxoid to form combined prophylactics for the immunization of man against diphtheria and whooping cough or these two diseases and tetanus simultaneously.

Schick Toxin

A Schick dose of toxin is that amount which causes a reaction when it is mixed with 1/1,250 unit of antitoxin and injected intracutaneously into a guinea-pig but does not cause a reaction when mixed with 1/750 unit; further, 1/25th of a Schick dose, without antitoxin, shall cause a reaction and 1/50th shall cause no reaction. In other words, a Schick dose of toxin is equivalent to 1/1,000 unit of antitoxin and contains 25 m.r.d.

In practice, toluene-preserved samples of toxin are set aside in the cold room and allowed to 'lager' for a year or more until toxoiding and destructive processes have become stabilized. They are then titrated and one containing 25 m.r.d. per Lr/1000 dose is chosen.

The Schick test is much more of a qualitative than a quantitative method of measuring immunity. People with 0·004 unit or more of antitoxin per ml. of serum will not react whereas those with less will react. But as at least 0·04 unit per ml. of serum is necessary to prevent an attack of diphtheria, nothing definite can be said of the 'real' immune state of a Schick-negative person. The test shows that he has some degree of immunity but not how much.

It is essential to inject the amount of Schick toxin prescribed by the manufacturers and not an amount that will produce a bleb in the skin, because this may be much less than the correct dose. A control injection of heated toxin must be given to pick out pseudo-reactors.

The test has its main application in mass survey when some idea of the immune state of a community is desired.

LABORATORY ASSAY OF PROPHYLACTICS

If every laboratory in the world used the same strain of *C. diphtheriae*, if this never varied in its metabolism, if the medium used was completely reproducible, and if all other conditions—temperature, period of incubation, method of preparing FT, purification etc.—were the same, the necessity for assaying a prophylactic for antigenicity would hardly arise. But this ideal state of affairs does not prevail.

In these laboratories, toxin is prepared weekly and, whilst the analysis of the culture medium varies only slightly from batch to batch, it does vary. Again, the Lf of each toxic filtrate is not the same, nor is the chemical composition of every FT. Although each purified toxoid contains between 1,800 and 2,000 Lf/mg. PN and the results of assays of different batches of PTAP for antigenicity are not, statistically, significantly different, it must be realized that the Biological Control Laboratory is not legislating for one laboratory only but for any that may issue a diphtheria prophylactic.

The test at present in force is a 'minimum requirements test', i.e. one that demands a specified minimum immunity in a specified number of guinea-pigs immunized with one or more specified doses of prophylactic. In the Institute, 10 or more guinea-pigs receive, subcutaneously, 1 Lf of PTAP or ADF in 0·5 ml. of $AlPO_4$ suspension containing 2·5 mg. of $AlPO_4$. This amount is again injected 28 days later and, 14 days after this, blood is removed from each animal. The antitoxin content of each serum is assayed at the Lr/1000 level in guinea-pigs and the geometric mean of the 10 or more values obtained is worked out. No prophylactic would be issued unless this value was at least 2 units per ml. of serum but, in practice, it is always between 5 and 10.

This is a satisfactory test—up to a point. It shows, for example, that a particular prophylactic is a satisfactory antigen when assayed in Johannesburg, in summer, in in-bred guinea-pigs fed on a good diet and housed in good conditions. But would the same result have been got if it had been tested in winter, in 'mongrel' guinea-pigs, badly fed and housed in another part of the world? In other words, it is possible that a prophylactic, passed for issue in South Africa, would be rejected in another country.

Little or no difficulty is encountered in titrating an antitoxin, because it can be compared under identical conditions with an international standard antitoxin of given value. International bodies are working on the preparation of a standard antigen with which an unknown prophylactic could be compared. When such a standard is available, the country, season, animal and food will play no part because whatever prevails for the prophylactic will prevail for the standard.

However, work being carried out in these laboratories indicates strongly that, where particulate prophylactics are concerned, many difficulties will have to be overcome before a generally acceptable method is devised. For example, will the test demand 1 or 2 injections, how many Lf will be used per injection, will the primary stimulus be larger or smaller than the secondary, and will the test involve the immunization of groups of guinea-pigs, the first with xLf the second with $x/2$ Lf and the third with $x/4$ Lf?

If a 1-dose method is adopted, it might lead to the rejection of a prophylactic that would pass easily by a 2-dose test. If 2 doses are stipulated, other difficulties arise. If the Lf dose is very small, it is possible that some eminently satisfactory prophylactics would fail and, if large, that some weak antigens would pass. Although some of the work going on is, possibly, of more academic than practical value, it is right that it should continue until a fully satisfactory test is worked out. The laboratory worker wants to prepare, and the physician wants to use, the best product available.

PREPARATION OF ANTITOXIN

The horse is the most important single factor involved in antitoxin production because, as producers, some animals are good and others bad. Naturally, the most suitable toxin, size of dose and spacing of injections, as shown by experience, are used, but a 'bad' horse cannot be turned into a 'good' one

by, for instance, injecting double the usual amount of antigen into it.

A toxin made in a pancreatic digest of ox flesh (see under 'Preparation of Toxin') is unsuitable because, in the large doses required for hyperimmunization, it upsets the animal and causes large swellings. Martin's broth, a peptic digest of pig's stomach and beef, is suitable because toxin made in it is reasonably well tolerated and it produces the desired result in most horses, viz. a high-titre antitoxin.

Each horse that enters a serum-production stud must pass the mallein and tuberculin tests (the last case of glanders was seen in South Africa in 1923 and tuberculosis is rare in the horse). Each horse's serum is then titrated for the presence of 'normal' diphtheria antitoxin by the intracutaneous method and only those horses with at least 0·1 unit per ml. are allocated to the diphtheria section of the stud. Among the last 100 horses entering the stables 14 were found to contain 1 or more units per ml. of serum, 41 between 0·1 and 1 unit and 45 less than 0·1 unit. The presence of 'normal' antitoxin has no connection with recovery from clinical diphtheria because the horse is not susceptible but is due to small, repeated, stimulations by the toxin of *C. diphtheriae* which may be found in abrasions, wounds and mucosae.

The 'natural' immunity of a horse provides a flying start to hyperimmunization. There is no need to 'prepare' the horse by 2 or more injections of APT and to wait for 6-12 months before starting a course as must be done when horses have to be hyperimmunized against tetanus toxin because 'normal' tetanus antitoxin is found only rarely in horses, if at all.

Some laboratories start immunizing with FT and use unmodified toxin only in the later stages, but in this Institute toxin is used from the start. The first few doses are small and are given daily until an amount of 5 ml. is reached. Thereafter, increasingly larger amounts are injected subcutaneously, thrice and later twice weekly until the dose is 300 or even 400 ml. According to the antitoxin response and physical condition of the animal, this primary course lasts 4-6 weeks. When a test shows that the serum antitoxin titre is 1,000 or more units/ml., 20 litres of blood are removed, (2×10 litres with a 4-day interval between bleedings) from the jugular vein into citrate solution. The horse is rested for 4-5 weeks, undergoes a rapid re-immunization lasting 10-14 days, is bled, is again rested and so on until its serum-antitoxin titre falls to 800 units/ml. or less. This happens at any time between the 5th and 15th course.

Concentration and Purification of Antitoxic Plasma:

To plasma containing 0·3% cresol and diluted with 2 parts of water, enough pepsin is added to make the concentration 0·25% and the pH is brought to 3·2. After the mixture has stood for 40 minutes at room temperature, ammonium sulphate, to 14%(w/v), is added and the pH is adjusted to 4·3. The precipitate that forms after heating at 55°C for 1½ hours is discarded and ammonium sulphate to a total of 31% (w/v) is added to the filtrate at pH 7·4. The precipitate is collected on filter pads, is pressed, and is dialysed in Cellophane bags for 6 days against cold running water.

The concentrated antitoxin globulin solution, preserved with 0·3% cresol, is clarified and diluted with 0·3% cresol-salin. to contain about 4,800 units per ml. as ascertained by the flocculation test. A preliminary *in vivo* titration is then carried out intracutaneously and sufficient cresol-saline is added to bring the value to 4,500 units/ml. The antitoxin solution is then sterilized by pad filtration and another intracutaneous test carried out to confirm that it contains at least 4,400 units per ml.

Sterility tests in culture media and safety tests (5 ml. subcutaneously in the guinea-pig and 1·0 ml. in the mouse) must be passed before the refined antitoxin is put into ampoules. The unitage of a few of these is checked *in vitro* and *in vivo*. Sterility and safety tests of the contents of ampoules taken at the start, the middle and the end of the filling process must be passed. The antitoxin is then ready to be issued.

Antitoxins may be 'avid' or 'non-avid', i.e. they vary in their ability to form a firm union with toxin. The unitage assigned to a non-avid antitoxin by the flocculation test will be higher than that obtained by an *in vivo* titration (L+ or Lr type of test) because when the toxin-antitoxin mixtures are diluted in body fluids after injection into a guinea-pig, they tend to dissociate. Thus, more antitoxin is required to neutralize the test dose of toxin in the animal than is needed for the same amount of toxin in the flocculation test. Biological control laboratories rightly demand the animal test as the method of titration.

In these laboratories, the *in vitro/in vivo* ratio of every diphtheria antitoxin issued is about 1, because each batch is made from the pooled sera of at least 20 horses; the effect of the possible presence of one or two non-avid sera is thus minimized.

The increase in purity obtained by salting-out methods (ammonium or sodium sulphate) is only about 50% whereas it is about 300% by the pepsin-digestion process (units of antitoxin per gram of protein). Pepsin splits the antitoxin-containing globulin molecule into two, and renders that portion of it that is poor in antibody heat-coagulable in the presence of ammonium sulphate. This part is discarded in the first precipitate. Refined antitoxin is much less liable to cause serum reactions, either immediate or delayed, than natural serum or sulphate precipitates.

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THE PSYCHOPATH AND THE MENTAL HOSPITAL*

M. GINSBURG, M.D.

Physician Superintendent, Fort Napier Hospital, Pietermaritzburg

To every mental hospital in any part of the world cases of psychopathy, or rather sociopathy, are admitted with regular monotony. These cases invariably, in one way or another, cause considerable inconvenience and interference with the welfare of the patient-body as a whole. The disrupting influence of the psychopath is well known to every hospital administrator and is a nightmare to both medical and nursing staff. This is due either to their constant demand for attention or to their ability to influence adversely the staff—and sometimes not only the nursing staff (see Minde's case, below). Whatever the cause, the result is detrimental to the treatment and well-being of the other patients.

Many of the sociopaths admitted to our hospitals come as the result of successful defence pleas supported by sympathetic psychiatric opinion. The evidence in these cases is usually given by psychiatrists or other medical men not associated with the hospital. When called upon to submit a report the hospital has to state that the individual is not mentally disordered within the meaning of the Mental Disorders Act.

Before mental hospitals began to change their character from purely custodial institutions or asylums to the hospitals we know today, they may have been suitable abodes for the sociopaths. But owing to the change in their character, which is still taking place, to send a sociopath to a mental hospital is not only inadvisable but wrong from several aspects. Thus, (*a*) with the replacement of custodial functions by therapeutic, the hospital is not equipped to detain him, and (*b*)—more important—is unable to offer him any hope of cure.

DEFINITION OF 'PSYCHOPATH'

The attitude accepted by most administrators is that psychopathy is a non-certifiable condition within the meaning of the Mental Disorders Act, and unless the psychopath is at the same time suffering from a frank mental disorder or defect he is not acceptable in a hospital. To avoid confusion and misunderstanding, the Mental Disorders Act was amended in 1957 and now omits 'Class VI', which catered for socially defective persons. This was the clause under which a defence plea for the admission of a psychopath as an individual suffering from mental disorder used to be made. Its elimination lessens the possibility that a psychopath or, for that matter, a socially defective person, will be admitted unless he is, in addition, a psychotic or a defective.

It is generally agreed that (1) the psychopath is free from all obvious signs and symptoms of a psychosis, but that he is more likely to develop a psychosis than his non-psychopathic brother, and that (2) although he is not certifiable he is as great a menace to society, if allowed a free rein, as any potentially dangerous psychotic allowed the same freedom.

Having discussed these general points, I now come to the definition of a psychopath. The definition which I find most acceptable is that of Dwight L. Moody,[5] of St. Catherine Hospital, Ontario, and is as follows:

> 'condition in which thought processes follow the normal patterns and uninterrupted sequence of mental activity but in which are found gross deficits and exaggeration of certain personality traits which lead to behaviour that is socially unacceptable. Subjects expressing this abnormal reaction to society are devoid of self-assessment, deficient in moral sense as judged by ordinary conventional standards, or in some instances or for other reasons merely incapable of putting simple rules of conduct into practice. Sociopaths are dispositionally biased and lack ability and desire for self-control, discipline and cooperation.'

More simply, Minde[3] defines it as 'emotional instability plus anti-social conduct'. However, I should also like to emphasize the point raised by Minde that occasional emotionability and anti-social conduct should not be accepted as sufficient for the diagnosis, particularly when associated with a crime, and that the condition should be persistent and substantial. Unless there is a long history of behaviour consonant with our ideas of psychopathy, the plea of mental disorder on the grounds of this condition should be avoided. These pleas occur most frequently in cases of murder and, though I fully agree that psychopaths do on occasions murder, it is a rare complication of this condition.

IN THE MENTAL HOSPITAL

I now come to the problem as seen by the mental-hospital authorities. Patients are admitted to a mental hospital as (1) certified or (2) temporary patients or as (3) voluntary boarders or (4) inebriates. In the first 3 groups it is extremely rare to find a psychopath. Amongst the inebriates they appear fairly frequently and in this group of psychopaths one seldom finds an individual who has been guilty of a serious crime. The majority, moreover, are alert. They never show the irrationality of the psychotic, they never accept the blame for their troubles, and they display all the symptoms so ably described by Cleckly.[1]

However, the psychopath as a problem as such is admitted to the hospital directly for observation under sections 27 and 28 of the Act when doubt has been expressed as to his sanity or when at the trial evidence has been led to show that he is insane. There are several possibilities concerning the individual sent for observation, and he may be placed in one of several broad groups:

I. Firstly, he may be suffering from a frank psychosis or defect. These psychotics or defectives are disposed of according to the nature of the crime. In the case of murder, they are invariably committed as 'Governor-General's Decisions' (GGD). In less serious charges the case is dealt with under section 6 of the Act and the individual is treated for his condition according to the nature of his illness. Should he recover, which is often the case, he is discharged and

*Presented at the South African Medical Congress, Durban, September 1957.

allowed to take his place in society as a useful unit once again.

I will not go deeply into the question of the discharge of a GGD patient. Suffice it to say that the duration of his stay following the episode depends on the nature of his illness, the time he has been recovered, the possibility of relapse, and the possibility of his future danger to society.

Seeing that satisfactory remedies are available, the hospital serves a useful function where this type of individual is concerned and is often of the utmost benefit; e.g. where the individual commits a crime while suffering from an infection or exhaustion psychosis, or the mother strangles her child while suffering from a recoverable form of puerperal insanity. Moreover, although less satisfactory, at least suitable custody can be given to the schizophrenic who does not respond to treatment. For him the chronic wards of the hospital will become his home and for the homicidal paranoiac the walls of the Fort at Bloemfontein will prevent his being a menace.

II. The second group sent for observation can be divided into two sub-groups, viz.:

(*a*) Those found not mentally disordered and who are not psychopathic.

(*b*) Those found not mentally disordered, but who are emotionally abnormal, with full insight and, as described by Clarke,[2] quite sane and in full touch with reality; amongst these are many of the pathological liars, sex deviants, misfits and those who commit crimes without sufficient object or motive.

As far as group (*a*) is concerned all that need be said is that any defence is worth trying when a case is hopelessly loaded, and here the mental hospital merely reports that he is neither mentally disordered or defective and does not enter into any argument about psychopathy.

Group (*b*) is a much more serious problem, largely because of the divided opinion on the certifiability of the psychopathic personality and, as already mentioned, the inability of the mental hospital to treat, cure or even give proper custodial care to such persons. To certify such a patient is undoubtedly tantamount to giving those prone to criminal acts a licence to commit crime at will, and I do not believe that such is the function of the mental hospital or the Mental Disorders Act.

To illustrate this point I should like to refer to two cases, one of which is the case of Dr. Minde, quoted with his kind permission. This case was detained at Sterkfontein Hospital.

Case 1

'Last year a psychopath came to Sterkfontein as a GGD patient, whom we shall call 'H'. He had been sent to us, rather reluctantly, by a judge who found him guilty of theft from a hotel room in Germiston.

Before H had been with us for many weeks we discovered that he had organized a dagga-smoking gang in his ward, which involved 6 or 7 of the patients. The dagga was obtained from a Native attendant he had bribed.

A little later he persuaded a young male nurse to leave the door of the ward open for a few minutes one night, and escaped, leaving a most artistically made dummy in his bed; it had completely fooled the night staff. He made his way to a town in the Northern Transvaal, where he posed as the representative of one of the big oil companies. He went to the leading hotel and was so plausible that the proprietor offered him unlimited credit. For the next few weeks he lived on the fat of the land, eating and drinking only of the best, and entertaining the local citizens royally.

There was an attractive receptionist working at the hotel, and H made violent love to her. He soon persuaded her to become engaged to him, quite ignoring the fact that he already had a wife and 2 children in Johannesburg. He gave her a fine diamond ring which he, of course, obtained on credit from the local jeweller. The night the engagement was announced he threw his biggest party yet. All the local gentry were there, and he would allow them to drink nothing but champagne. As he told us afterwards, he almost got writer's cramp signing I.O.Us. that night. He rounded off the evening by seducing his 'fiancee'. Early next morning, while everybody was sleeping off the effects of the party, he quietly slipped downstairs, and to misquote Milton, made for "fresh fields and pastures new".

The police caught up with him in a few days, and then he blandly told them: 'I'm an escaped GGD from Sterkfontein.' When he got back to us he appeared to have turned over a new leaf. He expressed the most unctuous remorse for his dastardly conduct, his behaviour was exemplary, and he even began to take an interest in work, a subject for which he had hitherto shown the most marked aversion. He so impressed the medical officer in charge of his ward that he began to think H quite an ill-used fellow, who had never had a real chance in life. He made himself very useful to the medical officer too, doing various odd jobs for him, and eventually offered to 'duco' his car, which needed a coat of paint badly. He rigged up quite an ingenious apparatus for this purpose, driven from the car's engine. This meant, of course, that he had to be given the car key—which is what he had been aiming at all the time. One fine afternoon, when all was peaceful and the doctor working in a distant part of the hospital, H quietly returned to his own ward, got into the room where the patients' private belongings were stored, picked out the best suit of clothes he could find, and drove off in the piebald car. He did not get far. At the entrance to Krugersdorp he collided with a delivery van and smashed up the car. With his usual luck he did not suffer a scratch, but got out in a towering rage and abused the other driver for ten minutes on end. Then he screamed: 'I'm going to telephone for the police', and stalked off, apparently bursting with indignation. That was the last seen of H, going round the corner with a parcel containing the suit still tightly tucked under his arm.

Glad as we were to be rid of him, we knew that the relief was only temporary unless we could do something about it. Our Superintendent, after consultation with the Commissioner for Mental Hygiene, recommended that H should be unconditionally discharged, and this was duly approved by the Governor-General in Council.

Sure enough, about 2 months later we received a letter from Durban Gaol stating that a man named H, who was serving a sentence of 10 months for fraud, claimed to be an escaped GGD from Sterkfontein. We replied happily that he had been unconditionally discharged from the operation of the Mental Disorders Act and could thus serve any sentence passed on him.

The moral of this story appears to be that when one has to decide whether a psychopath should be sent to a mental hospital, the extent of his mental abnormality is of much greater importance than his anti-social conduct.'

The second case, to whom we shall refer as 'S' shows the difficulties and uselessness of sending a psychopath to a mental hospital. Not only are we unable to treat him effectively under present conditions, but the protection which society demands is not possible, nor is the law able to play its role by removing such an individual from the public. In fact, certifying a psychopath makes the law and psychiatry farcial, as 'S' so well demonstrates.

Case 2

'S' was charged on 20 March 1953 with raping a female Native. From the evidence it would appear that the act, although spontaneous and unpremediated, was carried out by an individual who at the time of the crime was free from all psychotic symptoms. He took the opportunity which offered itself.

It would appear from the evidence that on the evening of 20 March S was travelling on a lorry with several Indians, when a Native female was given a lift. S gave her some gin to drink

and when the lorry stopped and she set off for her home, S followed her and, as shown in the evidence, assaulted her. That he was not disordered at the time of the preliminary examination is also obvious; the following is an extract from the court record: 'Accused states, 'Ek sê niks nie—ek sal my saak in die Hooggeregshof verdedig deur my prokureur'. (Translation: I refuse to comment and I will defend my case in the Supreme Court through my lawyer.')

After making these statements and arrangements, accused accepted short service and immediately applied for bail. What happened to this application I was unable to ascertain, but he was transferred to the local gaol on or after 24 April, on which date the preliminary examination ended. There is no evidence that the magistrate at the time even suspected mental disorder.

On 9 May 1953, S was sent for observation under section 27 of the Act, because he had been displaying symptoms suggesting mental disorder while in custody. The certificate of the District Surgeon which accompanied him was as follows:

> 'Gives irrelevant answers to questions, mumbles to himself, plays with pieces of torn material. He has torn his clothes. He laughs to himself. He keeps pacing the floor.'

The District Surgeon, who was well versed in matters psychiatric, in paragraph 2 of his 'Mental S.2', stated that when last observed several weeks previously S appeared normal.

It was also reported that S was destructive in his cell, and that he cut himself, was dirty in his habits, faced the wall talking to himself, and did not answer questions.

Unfortunately the exact date of onset of his symptoms cannot be established, beyond the fact that he was apparently not disordered at the end of the preliminary examination.

The report which followed his observation and on which he was made a GGD case is as follows:

> 'The patient's mental condition has shown little or no variation during the period of observation. He has remained disorientated in respect of time and place and too confused to give any useful information about himself or engage in relevant conversation. He often mumbles to himself, unheeding of what is said to him, and behaves restlessly, indifferent to his surroundings. Most of the intelligible mutterings made deal with army matters and he often says he has just seen the major or his own wife. He requires care, supervision and treatment.'

He was admitted to the Fort Napier Hospital on 18 June 1953. The report to the Commissioner for Mental Hygiene of 29 July 1953 is as follows:

> 'On admission from Town Hill Hospital, where he had been under observation, he was confused, manneristic and disorientated for time and place. Since admission until 6 July 1953 he had been grossly confused, unable to give any account of himself, and he was disorientated for time and place. On 6 July 1953 it is reported that he fell on his face in the airing court and from that date he has been mentally clear. At that day's interview he remains clear mentally but he has amnesia for events which occured over the past few months.'

He was diagnosed as suffering from psychopathic personality and hysteria. From the reports available, the differential diagnosis to be considered would appear to be (1) a Genser syndrome or (2) prison psychosis, or possibly even malingering. The suddenness with which he recovered after the fall on his face on 6 July is rather remarkable, to say the least. He maintained his condition and on 3 October 1953 he effected his escape.

Before his escape he denied that he knew anything of the crime and he stated he became confused because of financial and domestic worries. He also stated that he had been a patient in the Weskoppies Hospital, Pretoria, where he received electroconvulsive therapy. I could not trace any record of this in his file.

He was recaptured in Swaziland, where he was charged with theft and entering Swaziland without permission. He was found guilty of these charges but, as is so typical of these cases, traded on his being a patient at the Fort Napier Hospital, to which institution he was returned on 20 December 1953, only to escape again on 25 March 1954.

Report on his condition to the Commissioner for Mental Hygiene on his return is as follows:

> '. . . At present there are no symptoms of mental disorder. He appears to be a psychopathic personality with occasional attacks of hysteria. He is full of promises and good resolutions which should take him very far if they ever materialized.'

The note before his escape typifies his condition:

> 'He is very well behaved and cooperative. His conversation is rational. He has consistently denied the charge against him, and is anxious either to have his case reviewed or to be transferred from section 30 to chapter 1 of the Act.'

On 1 January 1954 S wrote a letter with a marked emotional appeal to the Minister of Health in which he pleaded his innocence with regard to the rape, but which demonstrates only too well the flaws in his claim of amnesia. The following extract is of particular interest.

'Ek het haar geklap met die vuis, want sy was brutaal nadat sy my drank gesteel het en uitgesuip het. Sy is oorlams en waarskynlik vanweë die drank wou sy eiegeregtig en selfs intiem met my word, vandaar die klappe aan haar toegedien.' Translation: I hit her with my fist, because she was brutal after she had stolen my liquor. She was cheeky and apparently as a result of the drink she became forward and wished to become intimate with me, and as a result the slaps followed.

From the time of his escape in 1954 until May 1957 he was not heard of again as far as we were concerned. Then he was charged with a petty theft and brought to the hospital by the South African Police. Fortunately, the police allowed him to go and his full discharge from the operation of the Act was subsequently obtained. Other points of interest in this case are that he had been known to the police since 1941, and that he was alleged to have been a prisoner of war.

This case, like that of Dr. Minde's, reveals the great difficulties encountered in detaining such an individual and, what is more important, how he has successfully thwarted the course of justice. Suffice it to say that though we must accept that he was disordered for a short period while he was in gaol, he evaded his just punishment and his removal as a potential danger.

Case 3

The patient 'W', to whom I shall refer briefly, has been in and out of mental hospitals and police lock-ups since 1940. She is well educated and went to the best schools. She has the unhappy knack of being able to play off patients against staff and staff against staff and by her knowledge of the Mental Disorders Act is able to exert such pressure on the courts that an investigation was ordered into the legality of her certification when she was in as a certified patient. Her ability to disrupt the welfare of the patient body is so well known that her possible re-admissions are viewed with trepidation. She has been known to break windows deliberately and cut her arms so that she could get the Medical Officer out of bed at midnight to stitch her wounds.

From such cases as the foregoing, there is little doubt in one's mind of the impossibility of detaining the aggressive psychopath in a mental hospital. Not only does he interrupt the welfare and treatment of patients who are capable of recovery, but he disrupts even the peaceful serenity of the chronic dement. He creates an unbearable and unnecessary tension among the staff, who unfortunately, like many others, are nowadays working at two-thirds or half strength. Moreover, these people must be a soul-destroying nightmare to the police; no sooner have they completed their task and got the psychopath safely tucked away in a mental hospital than he makes his escape, society is again exposed to his criminal propensities, and the search starts all over again.

TREATMENT

But although these people are not suitable as patients in a mental hospital, it cannot be denied that they are sick or

handicapped in much the same way as the psychotic or defective. The only question is whose responsibility they are. It is difficult to disagree with Checkley when he refers to the emancipation of the psychotic in his book *The Mask of Sanity*. Here he describes the work of Pinel, and mentions that it took a very long time before any active treatment was instituted for psychotics. His view is that a similar step is necessary for the psychopath, even though it may be a considerable time before successful treatment is available.

In the disposal of the patient sent for observation it is necessary to be particularly careful before diagnosing him as a psychopath. All other possibilities must have been excluded, amongst which are early schizophrenia, epileptic conditions, high-grade mental deficiency, hysteria, alcoholism, post-traumatic conditions, drug addiction, and even a hypomanic state; and—perhaps even more important—it is necessary to differentiate the psychopath from the 'normal' criminal. In Case 2 the possibility first of schizophrenia and then of hysteria was seriously considered at the time of his admission. Case 3 has on occasions been certified as suffering from hypomania. But on considering the history the diagnosis as a rule becomes obvious.

One is left with little doubt that unless special facilities are provided for the care of psychopaths, a psychopath found guilty of a crime is much more satisfactorily placed in prison than in a mental hospital; though one is left with the unpleasant feeling that we are then placing on the prisons a responsibility which is entirely medical. I know of no countries where such special provision is made for psychopaths except Denmark and Britain.

RECOMMENDATIONS

The opinion I venture as a solution to the problem is that an institution should be provided where such patients can be placed and cared for and where they can receive whatever treatment is considered advisable. A law, known possibly as the Psychopath Act should be enacted giving powers to detain such persons. The decision of placing a psychopath in a hospital should rest with a judge after consideration of both the record and the medical evidence. The sentence should be indeterminate, and discharge should only be considered after agreement by a board of psychiatrists, and should at first be strictly subject to conditions approved by a board of control comprising medical, legal, police and lay representatives.

An Institution of this kind, known as the Psychopathic Prison, has been established at Herstedvester near Copenhagen, Denmark. It is the first sign of the light for a dark and dismal condition.

The functioning of this hospital is described by Stephen Taylor,[4] who points out that the Danes have recognized that hypocritical matrimonial defrauders, brutal child violators and dangerous incendiaries are fit subjects not for punishment but for treatment, 'though treatment to be effective must be a stern discipline'. He discusses the Danish criminal law of 1930, of which section 16 deals largely with recognizable certifiable conditions as we know them, and section 17 with mentally abnormal criminals not covered by section 16. Section 17 covers those who at the time in point were suffering from some more permanent upset, but could not be said to be irresponsible; it also deals with sexual abnormality. Once a person is certified under section 17 he may be sent to an ordinary prison if he is likely to benefit from punishment, or he may be sent to a mental hospital, a home for inebriates or a psychopathic prison. If he is sent to a psychopathic prison the sentence is indeterminate.

Severe as this may seem, Taylor states that the authorities at Herstedvester claim a 50% cure-rate. He foresaw that many undiagnosed psychopaths would be found in prisons or mental hospitals without hitherto having been diagnosed, and therefore that adequate provisions would be necessary for their transfer to the psychopathic prison.

I myself approve of the idea of a psychopathic hospital but I dread the possibility of having anything to do with such an institution. Strange as it may seem, however, the experiences at Herstedvester have been contrary to what most of us would have anticipated. The findings of Dr. G. K. Stürup, the Superintendent of the prison, quoted by Taylor illustrate the point:

> 'At Herstedvester for a number of years we have had from 120 to 180 psychopathic detainees and prisoners, and they have caused considerably less trouble than when they were scattered over various hospital wards and prison wings among more ordinary prisoners. Here the widely divergent personality types hold each other in check, the staff gradually get sufficient training and experience, and modes of treatment can be laid down giving comparatively fixed limits to the individual displays that may occur. He that does not work will not earn money and so must go without tobacco, newspapers, and cakes on Sunday, just as in ordinary life. These are small things, but it is quite unpleasant to watch one's more industrious fellows reaping the benefits of their diligence.'

All the patients, as far as possible, are on piecework. They can earn up to 10s. a week, of which one-half must be saved.

The prison itself is described as being more like a modern mental hospital and having all the facilities which go to make such a hospital. Section 34 of our Mental Disorders Act permits of the transfer of psychiatrically sick prisoners to a mental hospital; as mentioned above, mental disorder develops in the psychopath more frequently than in his normal brother. Herstedvester have seen to it that facilities exist for dealing with this contingency.

Besides Herstedvester, I have heard of only one other institution which caters entirely for psychopaths and that is the Belmont Industrial Unit in Britain.

The answer to the psychopath problem would appear to me to have been given by Herstedvester; and I believe the time has come for us to consider the development of a similar scheme, with the object which Taylor states in expressing the opinion of Dr. Stürup: 'It is not a matter of making criminal psychopaths into normal people, but of making them into nice psychopaths who are capable of adapting themselves to ordinary life'.

This article is published with the kind permission of the Deputy Commissioner for Mental Hygiene, Dr. B. P. Pienaar, to whom my thanks are due.

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HYPEROSMOLARITY IN INFANTILE GASTRO-ENTERITIS

M. D. BOWIE, B.Sc., M.B., CH.B.

DAVID MCKENZIE, M.MED. PATH.

J. D. L. HANSEN, M.R.C.P., D.C.H.

Departments of Child Health and Pathology, University of Cape Town, and Red Cross War Memorial Children's Hospital Cape Town

Gastro-enteritis is a condition frequently associated with an excessive loss of both electrolytes and water from the body. The loss of water and electrolytes may be in proportion to the relative concentrations found in the body fluids or the loss of water may be proportionately greater or less than that of the electrolytes. It follows that states occur in which the concentrations of the serum electrolytes are raised (hyperosmolarity) or lowered (hypo-osmolarity). It should be noted that although the serum concentrations of electrolytes are raised in hyperosmolarity, there is still a total deficit of electrolytes. The serum figures merely reflect the excess water loss over that of electrolyte.

A varied terminology has been used to describe the condition of hyperosmolarity. Hyperelectrolytaemia, hypertonic dehydration and hypernatraemia are the terms most commonly encountered. Hypernatraemia is used because sodium salts constitute 90% of the osmotically active particles in the extracellular fluid compartment and the serum-sodium concentration is the most accurate and easily available method of measuring the tonicity or osmolarity of the body fluids. The accepted range of normal for serum-sodium concentration is 130-150 mEq./l. and any serum-sodium figure above 150 mEq./l. is considered to fall within the hyperosmolar group.

American workers[1-3] have attempted to lay down clinical criteria for the recognition of this syndrome of hyperosmolarity. In their series the great majority of cases occurred in infants under 1 year of age, and prematurely born infants appeared to be more prone to develop the condition. About 20% of infants with gastro-enteritis and dehydration presented with this syndrome. The gastro-enteritis was usually severe, with anorexia and vomiting. A history of severely curtailed fluid intake was often obtained. Some of the infants had in error received either hyper- or isotonic salt solutions in oral replacement therapy.

Clinical examination often showed minimal signs of dehydration and the skin felt doughy or sclerematous. Neurological signs were common and a preliminary diagnosis of a central nervous lesion of one type or another was often made. The neurological signs varied from lethargy to coma and convulsions. A common finding was increased muscular tone, occasionally to the extent of generalized rigidity and opisthotonus. Examination of the CSF showed an increase of proteins without pleocytosis. The serum showed high sodium and chloride concentrations but in addition calcium levels were lowered in a significant number of cases. Most cases were acidotic, with a lowered serum bicarbonate; hypopotassaemia and high blood-urea levels were frequent. Finally the mortality rate was higher in infants with hypernatraemia.

Gastro-enteritis is very commonly encountered in paediatric practice in South Africa.[4, 5] It was felt it would be of interest to ascertain the incidence of hyperosmolarity in cases seen in this unit. The following is a preliminary report of this investigation.

MATERIAL AND TECHNIQUE

During the first 7 months of 1957 (January-July) 89 cases of acute gastro-enteritis were admitted to the non-European wards of this hospital. Only the most seriously ill children could be admitted because of the limited number of beds. Serum-electrolyte studies were carried out in 39 of the 89 cases. In 32 of these the serum electrolytes were estimated on admission, and in the remaining 7 the estimations were done during relapses while in the ward. There was a random selection of cases in which electrolyte studies were done and the mortality of this group compares very closely with the general mortality (Table I).

TABLE I. NON-EUROPEAN CASES OF GASTRO-ENTERITIS ADMITTED

	Cases	*Deaths*	*Mortality*
Total	89	19	21%
Electrolyte group	39	8	20·5%

Blood was collected by internal jugular venepuncture. Serum sodium and potassium were estimated with an 'Eel' flame photometer. Serum chlorides were estimated by the method of Schales and Schales[6] and the carbon-dioxide combining power by the manometric method of Peters and Van Slyke.[7]

RESULTS

Frequency

The 32 cases in which serum-electrolyte estimations were performed before any treatment was instituted at the hospital (either in the ward or in the out-patient department) were further analysed (Table II). From this table it can be seen that 11 cases, i.e. one-third of the infants admitted to the hospital with gastro-enteritis on whom electrolyte studies were done on admission, exhibited hyperosmolarity.

TABLE II. CASES IN WHICH OSMOLARITY WAS ESTIMATED BEFORE FLUID REPLACEMENT

Osmolarity before any fluid replacement	*No. of Cases*	%
Hypo-osmolarity (serum-Na concentration < 130 mEq./l.)	7	22
Iso-osmolarity (serum-Na concentration 130-150 mEq./l.)	14	44
Hyperosmolarity (serum-Na concentration > 150 mEq./l.)	11	34

In addition 8 cases of hyperosmolarity were found either during relapses or after the patient had received a subcutaneous infusion or intravenous fluids. This gives a total of

TABLE III. CASES SHOWING HYPEROSMOPOLARITY

Case No.	Na mEq./l.	Cl mEq./l.	K mEq./l.	CO_2 mEq./l.	Blood Urea mg.%	Blood Ca mg.%	Special Features
1	169·0	143·5	6·88	7·8			* Gangrene of extremities.
2	159·0	130·7	9·4	7·5	250		* Sagittal sinus thrombosis.
3	170·0	139·4	5·55	7·1		8·9	Convulsions—responded to I.V. Ca gluconate.
4	204·0	158·5	10·0	20·8	149·0		Recovered uneventfully.
5	159·0	135·7	2·9	5·7			Salmonella infection.
6	165·0	129·8	4·98	22·7	137·5		Convulsions and generalized tremor.
7	154·0	113·8	3·4	—	113		Salmonella infection. Generalized rigidity.
8	152·0	112·0	6·6	5·1	52·6		Unconscious on admission.
9	156·0	133·7	4·7	11·0	42		Semiconscious on admission.
10	157·0	119	4·35	19·7	71·5		Associated pneumonia.
11	165·0	137	5·2	16·2	161		Uneventful recovery.
12	161·0	131	6·6	—	160		Neck rigidity. Cerebral cry. CSF normal.
13	163·0	134·2	6	13·2	46·5		No history of diarrhoea.
14	151·0	120·5	5·75	17·9	44	9·01	Convulsions.
15	154·0	116·8	4·5	14·8			Uneventful recovery.
16	163·0	118·8	4·15	24·2	65·5	9·9	* Sigmoid and transverse sinus thrombosis.
17	163·0	126·0	8·7	15·9	61·0		Athetoid movements. Air enceph.—cerebral atrophy.
18	167	137·6	8·3	—	52		Shigella infection. Convulsions.
19	156·0	101·0	3·5	34·0			Uneventful recovery.

* Cases that died. All other cases recovered.

TABLE IV. CASE WITH HIGHEST SERUM SODIUM (CASE 4)

Time	Na mEq./l.	K mEq./l.	Cl mEq./l.	CO_2 mEq./l.	Blood Urea mg.%
On admission	204·0	10·0	158·5	20·8	149·0
3 hours after admission	186·0	6·12	151·4	—	119·1
24 hours after admission	156·0	4·98	129·0	21·5	50·5

19 of 39 cases (i.e. one-half) showing hyperosmolarity at some time during the course of the gastro-enteritis. (Table III).

The highest serum-sodium figure was 204 mEq./l. found on admission in case 4 (Table III). In this case the serum electrolytes were estimated on admission, 3 hours after intravenous therapy commenced, and 24 hours after admission, and the figures show a rapid and satisfactory response to intravenous therapy (Table IV).

CLINICAL OBSERVATIONS

Skin Texture and Dehydration. All the cases of gastro-enteritis admitted to the ward were severely dehydrated on clinical examination, and this was confirmed by the weight gains observed after intravenous therapy. In only 3 cases of the hyperosmolaric group was a 'rubbery thick' texture of the skin noted, and this sign was noted in 1 case in which the serum values were normal.

Signs of Neurological Dysfunction. It is our impression that signs of neurological dysfunction are more marked in the hyperosmolaric group than in the non-hyperosmolaric group. This impression is supported by the percentage of cases in which either convulsions occurred or lumbar puncture was performed because of suspected CNS involvement. Of the non-hypernatraemic group 8% had convulsions and 12·5% were lumbar punctured. Of the hypernatraemic group 26% had convulsions and 31% were lumbar punctured. No rise in CSF protein was noted in this series (in contradistinction to the findings of other workers). In one of the hypernatraemic group an air encephalogram after recovery showed marked cortical atrophy, but this may well have been present

Fig. 1. Case 1. Gangrene of both feet (the fingers were also gangrenous).

before the illness. Rigidity and increase in muscle tone were not common clinical findings.

Biochemical Changes (Table III). Practically all the hyperosmolaric cases were acidotic, with a high blood urea (but so were the cases which were not hyperosmolaric). Serum potassium was raised in 10 cases and normal or lowered in the remaining 9. Serum calcium was estimated in only 3 of our series.

Mortality and Cause of Death. Of the 19 infants who became hyperosmolaric at some time during their illness, 3 died, a mortality of 16%. This compares favourably with the general mortality rate, but owing to the smallness of the numbers is probably not significant. The post-mortem findings in the 3 fatal cases which died are of considerable interest, showing that all 3 died with thrombosis:

1. Case 16. Thrombosis of the right transverse and sigmoid sinuses with infarction of the greater part of the right cerebral hemisphere.

2. Case 2. Thrombosis of the superior sagittal sinus with extension into the cortical veins.

3. Case 1. Gangrene of the feet and hands with thrombosis of the peripheral capillaries (Fig. 1).

DISCUSSION

As can be seen from the data presented, the clinical recognition of hyperosmolarity in infantile gastro-enteritis is difficult, and in our experience the diagnosis is usually biochemical rather than clinical.

It was impossible to extract any valid details of oral fluids given at home before medical advice was sought, but it is our impression that those showing the hyperosmolar state (excluding those essentially iatrogenic) had a short history of explosive diarrhoea and vomiting. This is in contradistinction to the non-hyperosmolar cases, in many of which there was a history of chronic or recurrent diarrhoea. In 3 cases showing the hyperosmolar state no history of diarrhoea or vomiting was elicited, and they presented simply as dehydrated acidotic infants. It was only after admission to the ward that they began to pass loose, offensive stools.

In this small series there were no clinical features on which a diagnosis of the hyperosmolar state could be made with conviction. Perhaps the most important outcome of this investigation is the realization that the hyperosmolar state is relatively common; 50% of infants with acute gastro-enteritis admitted to the ward and on whom electrolyte studies were performed were affected in this way at one time or another. Where gastro-enteritis is associated with hyperpyrexia, hyperventilation or severely curtailed fluid intake the possibility of hyperosmolarity developing is considerably enhanced by the excessive water loss.

Convulsions, disturbances in consciousness and other neurological signs were observed in one-third of the cases with hyperosmolarity, 2-3 times more commonly than in the non-hyperosmolar group. A doughy or sclerematous texture of the skin was found comparatively seldom and may also be found in the non-hypernatraemic infant. The disparity between this negative finding and that of other workers[1-3] may be accounted for by the fact that most of the children seen here show evidence of malnutrition and have little subcutaneous fat to give the rubbery feel to an examining hand. Serum-electrolyte estimations of course give the diagnosis immediately and it is of interest to note that the serum-chloride level is often relatively higher than that of sodium.

The 3 deaths were due to thrombosis, possibly related to haemoconcentration and increased viscosity, but the exact significance of this finding requires further investigation.

From the practical point of view treatment of gastro-enteritis must aim at replacing water and electrolytes. Intravenous therapy is the most effective method of doing this in a grossly dehydrated infant. The same general principle applies to infants exhibiting the biochemical features of hyperosmolarity. The serum levels of the electrolytes are high but it must not be forgotten that there has been an absolute loss of electrolyte and that both water and electrolyte must be replaced. If water alone is replaced the sudden drop of serum electrolyte concentrations may lead to the symptoms of water intoxication. Correction of the metabolic disturbance should be gradual, fluids being used with a low concentration of sodium and chloride. Half-normal saline (or half-strength Darrow's solution) containing 2½% dextrose is a very useful combination and should always be used where serum-electrolyte concentrations are high or unknown. Isotonic solutions of saline, i.e. normal saline, should not be used for any length of time except in the immediate treatment of shock and then should be followed by the administration of equal amounts of 5% dextrose water. Correction of acidosis with M/6 lactate should be cautious and if possible avoided, for this is equivalent to giving normal saline. In all except the grossest cases of acidosis due to gastro-enteritis, correction of dehydration, replacement of electrolyte loss, and the concomitant restoration of renal function, will lead to correction of the acidosis. Elimination of infection with antibiotics is of course an important part of the treatment of gastro-enteritis.[8]

SUMMARY

Hyperosmolarity is defined and a brief résumé is given of the findings of various American authors.

The condition as it is seen at the Children's Hospital in Cape Town is described. Our experience demonstrates that it is difficult to diagnose clinically, but it is relatively common and should be suspected in any child with gastro-enteritis with convulsions, a 'rubbery thick' texture of the skin, hyperpyrexia, or a short history with severely curtailed fluid intake.

Treatment should be aimed at replacing both electrolytes and water, but electrolyte replacement should be cautious, preferably with half-strength solutions.

We wish to thank Prof. F. J. Ford and Dr. I. Mirvish for clinical facilities, and Prof. J. G. Thomson and Dr. J. Mostert, Medical Superintendent, Red Cross Children's Hospital, for permission to publish. Thanks are also due to Mr. T. Turner for technical assistance.

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THE FINGER-PRINT SWEAT TEST

PINCUS CATZEL, M.B., B.CH., M.R.C.P.E., D.C.H.

Pretoria

In the United States and in England the estimates of the incidence of cystic fibrosis of the pancreas amongst children of European descent have varied from 1 : 1,000 to 1 : 10,000 (Lowe *et al.*,[1] Bodian[2]). If we assume a European population in South Africa of 2,500,000 then we expect to find between 250 and 2,500 cases in the country. In fact most paediatricians see only isolated cases. This means either that the incidence in South Africa is lower than overseas or that a large number of cases are being missed. With modern treatment a great deal can be done for these unfortunate children and every effort should be made to detect cases as early as possible before irreparable damage has been done.

I do not think that a case of cystic fibrosis has ever been described in a true Negro, but in Michigan, U.S., it is estimated that the incidence in Coloured patients is less than one-tenth of that amongst Europeans.[3] In South Africa there are far more pressing problems amongst the non-Europeans than cystic fibrosis and it is seldom that the disease is suspected. A widespread survey, however, would be of considerable academic value, particularly if whole families could be screened.

In this article there is no need to review the present knowledge of the disorder, because two excellent monographs and an extensive review have appeared in recent years.[2, 4, 5] It may however be of value to emphasize certain features of the disorder and the methods of diagnosis.

CLINICAL FEATURES

Cystic fibrosis of the pancreas is a generalized disorder of the eccrine glands characterized by abnormal viscosity of the mucus and an increased concentration of the electrolytes in the sweat. The disorder should be suspected under the following circumstances:

1. When a known case exists in a family all other members should be suspect, particularly infants and children. Sometimes cases are diagnosed in related families, e.g. amongst cousins.

2. Any case diagnosed as coeliac disease in which there is an associated chronic respiratory infection of any type, is a case of cystic fibrosis of the pancreas until proved otherwise. Today cystic fibrosis is probably commoner as a cause of steatorrhoea than idiopathic coeliac disease. All cases of steatorrhoea, therefore, which do not respond to a gluten-free diet should be suspected of suffering from cystic fibrosis and investigated accordingly. A feature of the steatorrhoea in cystic fibrosis of the pancreas is the extraordinarily foul and penetrating odour of the stools; mothers sometimes complain that it 'stinks the house out'.

3. Failure to gain weight associated with a voracious appetite. This paradox of an infant or child who 'eats as much as his father' yet fails to gain weight is fairly common in cystic fibrosis.

4. Chronic, repeated and recurrent respiratory infections, particularly those dating from infancy, are good grounds for suspecting the disease. Chronic dry, useless cough often occurs. Chronic sinusitis, asthmatic bronchitis and hoarseness of the voice are sometimes found. Some cases present as a 'second attack of whooping cough', the misleading feature being the paroxysmal type of cough which may occur.

5. All cases of staphylococcal pneumonia and empyema in infancy should be suspected of the disease.

6. Bronchiectasis, usually generalized and cylindrical occurs in advanced cases.

7. The disease should be suspected in all cases of neonatal obstruction due to abnormally viscid meconium (meconium ileus); 1 in 9 cases of cystic fibrosis present as meconium ileus.

8. Excessive sweating about the head in infancy may occur.

All grades of the disorder may be found. It may present in infancy as intestinal obstruction due to meconium ileus; in infancy as chronic diarrhoea or respiratory infections; or symptoms may not appear until the child is 5 or 6 years of age, when almost any combination of respiratory or alimentary symptoms may occur.

It is rare for these children to survive to adulthood, the majority dying before puberty, though in recent years an increasing number are surviving beyond this age. There is evidence that very mild cases may enter adulthood.[5]

DIAGNOSIS

The diagnosis can usually be suspected on clinical grounds, but the aid of the laboratory is essential to prove the diagnosis.[5] Until the advent of the sweat test, duodenal intubation and the analysis of the duodenal contents for viscosity and digestive enzymes was the only reasonably reliable test—and it called for special experience in the techniques involved; errors in collection and analysis are easily made. Examination of the stools for trypsin and for fats have not always been reliable. Indirect tests such as the vitamin-A absorption curve, sugar tolerance curve, chylomicron count, iodine excretion test and nitrogen balance tests may assist in the diagnosis but are fraught with error if not performed regularly. The sweat test however gives a high degree of reliability and is the subject of this communication.

Disordered Sweat Secretion

Until recently, as a result of Farber's work,[6] the most acceptable explanation of the aetiology of cystic fibrosis of the pancreas was that the disease was due to a general disorder of the mucus-secreting glands. Farber coined the term 'mucoviscidosis', i.e. 'viscid-mucus disease', and explained that the majority of symptoms were due to the presence of plugs of abnormally tough viscid mucus in the ducts of various organs. In the intestinal tract this resulted in meconium ileus in the newborn, in the pancreatic ducts it caused depression of pancreatic enzyme excretion into the duodenum; in the bronchioles it caused atelectasis, bronchial spasm, emphysema and secondary infection; and occasionally in the biliary tract it resulted in biliary cirrhosis.

In 1948, during a particularly severe heat wave, 10 children were admitted to the Babies' Hospital in New York with heat exhaustion. Of these cases 5 were known to suffer from cystic fibrosis of the pancreas. In subsequent years 2 further

cases developed heat exhaustion and the series were reported by Kessler and Anderson.[7]

The work was taken up by di Sant'Agnese, Darling and Perera,[8a, 8b, 8c] who measured the sweat electrolytes in 43 known cases of cystic fibrosis and in 50 controls. They found that there was a consistently greater excretion of sodium and chloride in the sweat in cystic fibrosis amounting to 2-4 times that of the controls. An increase in the potassium content was also found but it was not so marked. There was apparently no change in the volume of sweat excreted. The work was subsequently fully confirmed by Shwachman *et al.*[5] and by Webb *et al.*[9] However, the test was cumbersome and required at least half to one hour to collect a suitable sample. Shwachman *et al.*[10] therefore modified the test into a semi-quantitative screening test, the 'finger-print sweat test', which gave 99% reliability in their hands and was subsequently confirmed by MacFarlane *et al.*,[11] who obtained 100% results with it.

The Shwachman Finger-Print Sweat Test

The test is performed by *gently* and *lightly* pressing a finger or toe onto the test medium. The medium is made up as follows: 25 g. of pure agar is dissolved in 500 ml. of boiling water. When the mixture is melted 4·2 g. of silver nitrate is added and the mixture stirred. Then 2·5 g. of potassium chromate is stirred in. The hot, red mixture, which should be as clear as a bacteriological blood plate, which it resembles, is poured into Petri dishes and sealed with cellotape. Plastic containers can also be used.

The chloride on the finger bleaches the medium and the degree of bleaching is reported as 1, 2 or 3 plus. A 1+ reaction shows only slight bleaching and indicates a salt concentration of less than 60 mEq./l. A 3+ reaction is a deep white or yellowish-white colour change indicating a concentration of over 250 mEq./l. In between lies the 2+ reaction, which is easily distinguished with a little experience. In Shwachman's preliminary results in 140 known pancreatics, 138 gave a 3+ reaction and 2 a 2+ reaction; none gave a 1+ reaction. Of children with various diseases only 4 out of 103 gave a 3+ reaction. Amongst healthy infants there were no 3+ reactions. Of special interest is the fact that about 15% of parents of patients and about 15% of siblings have a 3+ reaction, a fact which may yet prove to be of great genetic interest.

MacFarlane *et al.*[11] reported the results in 54 cases of cystic fibrosis and 200 controls and obtained no false positives. In doubtful cases the hands were thoroughly washed and the cases retested at 5-minute intervals. All cases of cystic fibrosis became 3+ again within 20 minutes whereas none of the controls became 3+ within 20 minutes.

AUTHOR'S CASES

In South Africa we do not have the facility of collecting these cases within one large centre as has been done overseas. As a result many cases are missed. In my own practice I have picked up only one proved case in the past 2 years and have seen 2 further cases at the hospital, both unfortunately before the sweat test was available. Several other possible cases I have been able to disprove either by duodenal intubation or by means of the sweat test. I have been doing the finger-print test as a routine for some time, and it may be of interest to report my preliminary results.

Material and Method

Up to the time of writing, 230 children have been tested—42 European and 188 Bantu. Of the European children, 36 were tested by routine in my private practice, and 6 were seen at the Beatrix Street Hospital for possible pancreatic disease. All the Bantu children were seen at the non-European out-patient department; they presented with a variety of complaints—kwashiorkor, whooping cough, gastro-enteritis and respiratory disorders predominated. The ages ranged from 3 months to 12 years and the sexes were more or less equally distributed.

In all cases with a 2+ or a 3+ reaction the hands were thoroughly washed and the test was repeated after 20 minutes.

Results

Table I shows the results of the preliminary tests. Of the 230 cases, 37 gave a 2+ reaction and 7 a 3+ reaction. After the hands were washed all cases became 1+ except the known case of cystic fibrosis, which became 3+ again within 20 minutes (Table II). A note whether the day was hot or cool was made at the time of testing and the results are recorded in Table III. Our impression is that on very hot days one sees stronger reactions than on cool days, especially amongst Bantu patients, though a much larger series in a wider temperature range will be required to confirm this.

TABLE I. RESULTS OF PRELIMINARY FINGER-PRINT READINGS IN 230 PATIENTS

	+	++	+++	*Total*
European	31	8	3	42
Bantu	155	29	4	188
Total	186	37	7	230

TABLE II. RESULTS OF FINGER-PRINT READINGS 20 MINUTES AFTER WASHING OF HANDS

	+	++	+++	*Total*
European	41	0	1*	42
Bantu	188	0	0	188

* Known case of cystic fibrosis of the pancreas.

TABLE III. EFFECT OF TEMPERATURE ON THE RESULTS OF THE FINGER-PRINT TEST

	Cool Day				*Hot Day*			
	+	++	+++	*Total*	+	++	+++	*Total*
Eur. No.	25	5	2	32	6	3	1	10
%	78	15·6	6·4	100	60	30	10	100
Bantu No.	97	10	0	107	58	19	4	81
%	90·6	9·4	0	100	71·6	23·4	5	100

It is of interest that of 17 children with kwashiorkor only 2 gave a 2+ reaction and both were complicated by pneumonia—the rest were 1+ and often no visible change on the test plate was made by their finger-prints. Of 11 infants with gastro-enteritis none gave more than a 1+ reaction Most of the cases who gave a 2+ or a 3+ reaction on preliminary testing had respiratory infections, though a few cases were apparently normal.

DISCUSSION

Although the reasons for the increased electrolyte concentration in the sweat in cystic fibrosis of the pancreas are not

known, its occurrence has provided us with one of the most specific diagnostic tests known in medicine. It seems clear from the literature that the Shwachman finger-print test for chlorides, which was originally designed as a screening test, may prove to be an even more valuable diagnostic procedure than the original sweat test, which is cumbersome and requires skilled laboratory assistance in its performance. From my own small experience with the test I am convinced that it may safely replace duodenal intubation as a diagnostic procedure, particularly in areas where intubation is seldom done.

So long as the test medium is freshly made and checked against suitable controls, no difficulties should arise in its use, once one has gained a little experience in interpreting the results. I find that a good control is to apply one's own fingers one after the other onto the same spot, thus getting increasing bleaching on that spot. If one finger produces a slight change in the medium, then applying a second finger produces a 2+ reaction and a third finger a 3+ reaction. By this means one easily gains experience in reading the results.

In infancy, sweating is normally at a minimum and it is uncertain at what age it becomes sufficient to test. MacFarlane *et al.*[11] found a 3+ reaction as early as 4 weeks of age in an infant known to have cystic fibrosis of the pancreas. Normally infants do not produce even a 1+ impression until at least 3 months of age. Shwachman thought that the test was satisfactory after the age of 3 months.

Owing to the uncertainty at what age the finger-print chloride test becomes positive, an additional screening procedure is useful. The stool trypsin test, also developed by Shwachman,[12] may be used in the first year of life. Three negative stool trypsin examinations in the 1 : 5 or 1 : 10 dilution should be considered sufficient evidence for the tentative diagnosis of cystic fibrosis. The test can be confirmed after the age of 3 months (perhaps sooner) by the finger-print test, thus giving one a double check.

In our short series no false positive reactions were obtained in European or Bantu children. As was expected, cases of gastro-enteritis, most of which were severe, did not produce more than a 1+ reaction, probably because of the excessive loss of electrolytes in the stool. Whether electrolyte retention occurs in the oedematous tissues in cases of kwashiorkor, resulting in a compensatory 'shutting off' of electrolyte in the sweat, is to be further investigated in a larger series. The large number of 3+ reactions occurring in the preliminary tests on children with fever suggests an increased electrolyte excretion in these cases, but after washing the hands no more than a 1+ reaction occurs 20 minutes later. The test is obviously of no great significance in these cases and merely emphasizes the need to repeat the test after washing.

SUMMARY

1. Preliminary experience of the Shwachman finger-print test for chlorides in the sweat in routine practice is described in 230 children aged from 3 months to 12 years. No false positives were obtained.

2. The value of the test as a routine screening test and as a specific diagnostic test for cystic fibrosis of the pancreas is discussed.

3. It is suggested that during the first year of life the best results will be obtained by combining the Shwachman finger-print test with the examination of 3 consecutive stool specimens for trypsin.

I should like to thank Prof. J. G. A. Davel for his encouragement in this project and for giving me access to the cases under his care. Thanks are due to Dr. P. J. Pretorius and Dr. T. Dunston for reading the manuscript, and to Dr. P. J. Swanepoel, Superintendent of the General Hospital, Pretoria, for permission to publish.

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THE COLLEGE OF PHYSICIANS AND SURGEONS OF SOUTH AFRICA

An Extraordinary General Meeting of the College was held on Wednesday 5 March 1958 at 5.15 p.m. at the office of the College, 409 Medical Centre, Cape Town. Dr. A. W. S. Sichel was elected Chairman of the meeting. There being 15 members present and 42 proxies, the Chairman declared the meeting duly constituted.

Note was taken of the recommendations of the Annual General Meeting on 14 September 1957: (1) 'That the name of the College should be altered to "The College of Physicians, Surgeons and Gynaecologists of South Africa", (2) "That the lists for Associate Founders be re-opened".'

Name of the College

The following resolution was carried unanimously:

That subject to the approval in writing of the Minister being duly obtained, the name of the College be changed to 'The College of Physicians, Surgeons and Gynaecologists of South Africa', and that paragraph 1 of the Memorandum of Association of the College and the definition of 'The College' in Article 2 of the Articles of Association of the College be altered accordingly.

Associate Founders

The following resolution was carried with one dissentient:

That the following new sub-article (3) shall be substituted for sub-article (3) of Article 5 of the Articles of Association of the College:

'(3) An Associate Founder shall be a person:

(*a*) who applies for membership of the College within a period to be determined by the Council of the College in its discretion,

(*b*) who is found by the Council of the College to possess the qualifications entitling him to be registered as a medical practitioner in the Union of South Africa by the South African Medical and Dental Council and is found to be a person of good character and in good professional standing.

(*c*) who within the period determined by the Council of the College in accordance with the provisions of the preceding sub-article (*a*) complies with all the provisions of the Articles By-Laws and Regulations of the College and contributes a sum of not less than thirty guineas (£31 10s. 0d.) towards the founding or endowment of the College within such period as the Council of the College may fix, and

(*d*) whose name is duly enrolled and remains so enrolled as a member on the Register'.

FELLOWSHIP EXAMINATION RESULTS

The following candidates were successful in the Fellowship examinations of the College held at the University of Cape Town on 30 September and 2 and 3 October 1957.

Final Examination for the Fellowship of the College of Physicians of South Africa:

P. Markman, S. J. Saunders, L. Potgieter, I. B. Welsh.

Primary Examination for the Fellowship of the College of Surgeons of South Africa:

P. A. H. Knocker, H. Maisel, P. S. Willers.

Examination for the Diploma in Midwifery of the College of Obstetricians:

P. J. Turner.

The examiners for the Primary Fellowship in Medicine were: Prof. G. A. Elliott, Prof. F. Forman, Prof. A. J. Brink, Dr. M. M. Suzman, Prof. J. F. Brock, with Sir Russell Brain as overseas observer.

The examiners for the Primary Fellowship Examination were: Mr. Lee McGregor, Prof. L. H. Wells, Prof. J. Barnetson, Prof. A. W. Sloan, Prof. J. T. Irving, Prof. M. R. Drennan, Prof. C. F. M. Saint, Prof. J. G. Thomson, Prof. J. H. Louw, with Sir Harry Platt as overseas observer.

The examiners for the Diploma in Midwifery were: Prof. E. C. Crichton, Prof. James T. Louw, Prof. L. J. Te Groen, Prof. O. S. Heyns, with Mr. A. J. Wrigley as overseas observer.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

MAGAURAN'S ADAPTOR AND MOBILE TUBE SPATULA

Westdene Products (Pty.) Ltd. supply the following information:

Now available Magauran's adaptor (A) and mobile tube spatula (B), manufactured by Down Bros. Mayer and Phelps Ltd. The object of the device is to provide a Boyle's tongue plate which can be adjusted to suit patients of any size, from a small child to a big adult. From the distal part of the spatula a steel tube projects, to which a short intratracheal tube can be attached.

Fig. 1. Magauran's adaptor and mobile tube spaluta (W. H. B. Magauran, F.R.C.S.).

The small plate (C) which slides onto the tube spatula is used to widen the spatula for outsize patients. The adaptor can be suspended in such a manner as to give varying degrees of tilt to the tube spatula.

The device can be used with Draffin's bipods, or the Magauran triangular frame mentioned in the following reference: Lancet, 1957, **2**, 927 (9 November).

* * *

BENZACYL

Westdene Products (Pty.) Ltd. announce the introduction of Benzacyl and supply the following information:

Benzacyl is a PAS derivative which is almost free from the disadvantages of the original product. Like PAS, it is intended for the combined long-term treatment of tuberculosis.

Benzacyl is the calcium salt of N-benzoyl-p-aminosalicylic acid (calcium benzoyl-PAS, benzopas or BPAS). It was first thought that it acted by slowly releasing PAS in the body but it now appears as though this is not the case. This is shown by the fact that Benzacyl, which is almost completely absorbed from the bowel, is excreted in the urine largely as benzoyl-PAS with only small quantities of PAS, acetylated PAS and glycocolated PAS. The actual mode of action of Benzacyl is not known. It clearly differs from that of PAS because it is not inactivated by high concentrations of para-aminobenzoic acid. *In vitro* Benzacyl is 100 times less active than PAS but *in vivo* the two products are weight for weight equally effective. In actual practice however Benzacyl gives far better results because it has the following advantages over PAS:

1. Adequate blood levels are more easily maintained: Benzacyl is slowly absorbed and slowly excreted (peak blood levels are not reached until 3 hours after ingestion). Therefore it need be given only 3 times a day. PAS, because of its rapid absorption and excretion, should theoretically be given every 3 hours round the clock. Since in practice this is rarely done, consistent blood levels are not maintained with PAS as with Benzacyl.
2. Better absorption: Benzacyl tablets disintegrate almost immediately in water and complete absorption is therefore assured. PAS tablets have a much longer disintegration rate and in fact cases have been reported of the tablets passing right through the system and being excreted whole.
3. Better tolerated: Benzacyl does not cause gastric upset to the same extent as PAS. Few patients are unable to continue treatment because of this trouble. Two-thirds of the patients who cannot tolerate PAS are able to take Benzacyl without difficulty.
4. Better taste: Benzacyl is almost free from the most unpleasant taste which makes many patients refuse PAS.
5. Greater convenience: Four Benzacyl tablets need be given only 3 times a day in an average case, compared with the higher and more frequent dosage of PAS.
6. Less resistance: Resistance does not build up as readily to Benzacyl as to PAS. There is no cross resistance between the two drugs, so that Benzacyl may be useful in cases which have developed resistance to PAS.
7. Fewer allergic reactions: They rarely occur with Benzacyl as compared with the estimate of 4% of all cases treated with PAS.

Because of these advantages Benzacyl is gradually replacing PAS in many countries throughout the world. Originally introduced in 1948, its value has now been confirmed by many workers who suggest that Benzacyl will not only give a higher percentage of cures than PAS but will also reduce the hospitalization time for tuberculosis patients.

Benzacyl is available in bottles of 400 or 2,000 1·0 g. tablets and in bottles of 100 sachets each containing 4·0 g. of Benzacyl powder.

From the sole South African agents, Westdene Products (Pty.) Ltd., 23 Essanby House, 175 Jeppe Street, Johannesburg, or Cape Town, Durban or Pretoria branches.

* * *

TETREX INTRAMUSCULAR 250 MG.

Bristolabs (Pty.) Limited announce the introduction of Tetrex Intramuscular 250 mg., and supply the following information:

For the first time, Tetrex Intramuscular (250 mg.) provides a practical intramuscular-dosage form of tetracycline which produces and maintains antibiotic blood levels for 24 hours following

a single injection. This is possible because of the unique solubility properties of tetracycline phosphate complex, permitting maximal tetracycline activity for administration in minimal volume of injection. Extensive clinical experience has shown that such Tetrex injections are well tolerated with relatively minor discomfort.

Indications

For initial therapy and until oral medication (Tetrex capsules or syrup) can be given. Tetrex Intramuscular with Xylocaine is recommended in many common infections due to tetracycline-sensitive organisms. Indications for Tetrex Intramuscular include the following infections due to tetracycline-susceptible organisms:

Respiratory Tract Infection: Pneumonia, acute bronchitis, pharyngitis sinusitis, septic sore throat, whooping cough.

Urinary Tract Infections: Pyelonephritis, pyelitis, cystitis, prostatitis, urethritis.

Gastro-intestinal Infections: Bacillary and amoebic dysentry, bacterial diarrhoea, gastroenteritis.

Dermatologic Infections: Cellulitis, furunculosis, pustular dermatosis, acne.

Rickettsial and Viral Infections: Typhus fever, trachoma lymphogranuloma venereum, psittacosis.

Prophylaxis in Surgery and Obstetrics: Before or after surgery in unsterile fields.

Dosage

For the adult the usual minimum daily dose is 250 mg. administered once every 24 hours. Severe infections may require injections of 250 mg. at 12-hour intervals.

For children dosage should be adjusted according to body weight and conditions being treated. Clinical experience indicated that the daily dosage for infants and young children should be 15-25 mg. per kg. of body-weight up to a maximum of 250 mg. per single daily injection. Children of older and higher weight require at least 250 mg. per day, depending upon the clinical judgment of the physician.

Tetrex Intramuscular should be injected deeply into the gluteal muscle.

Presentation

Each vial contains 250 mg. of Tetracycline Phosphate Complex and should be reconstituted with 1·6 c.c. of sterile water or normal saline before injection.

Manufacturers: Bristol Laboratories Inc., Syracuse, New York.

S.A. Distributors: Bristolabs (Pty.) Ltd., P.O. Box 2515, Johannesburg.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY—ASSISTANT EDITOR

Applications are invited from medical practitioners for the post of Assistant Editor in the service of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale attaching to the post is £1,250×50—1,750 per annum, plus an annual cost-of-living allowance of £176 for single men and £352 for married men. The commencing salary will be determined according to experience.

The successful applicant must contribute to the Association's Superannuation Fund. He will also be expected to assume duty as soon as possible after appointment.

Applications must reach the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town, before 31 March 1958.

A. H. Tonkin
Secretary

Medical House
Cape Town
24 February 1958

VAKATURE—ASSISTENT-REDAKTEUR

Aansoeke word van geneeshere ingewag vir die betrekking van Assistent-Redakteur in diens van die Mediese Vereniging van Suid-Afrika, by die Hoofkantoor te Kaapstad.

Die salarisskaal aan die pos verbonde is £1,250×50—1,750 per jaar, plus 'n jaarlikse duurtetoeslag van £176 vir 'n ongetroude en £352 vir 'n getroude man. Die aanvangssalaris sal volgens ondervinding bepaal word.

Die suksesvolle kandidaat moet by die Vereniging se pensioenskema aansluit. Hy sal ook verwag word om so spoedig moontlik na aanstelling diens te aanvaar.

Aansoeke moet die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, bereik vóór 31 Maart 1958.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
24 Februarie 1958

MEDICAL ASSOCIATION OF SOUTH AFRICA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held at Medical House, 5 Esselen Street, Johannesburg, on 23 April 1958, at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House,
Cape Town.
21 February 1958.

MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word te Mediese Huis, Esselenstraat 5, Johannesburg, op 23 April 1958, aanvang 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis,
Kaapstad.
21 Februarie 1958.

PASSING EVENTS : IN DIE VERBYGAAN

Research Forum. The next meeting of Research Forum, University of Cape Town, will be held in the large A-Floor lecture theatre, Groote Schuur Hospital, Cape Town, on Wednesday 2 April 1958 at 12 noon. Speakers, Dr. A. Kipps and Dr. G. Selzer. Subject, Coxsackie Virus Myocarditis of the Newborn.

* * *

Dr. Jean Walker, lately of Cape Town, has joined Dr. John Leeming, of 415 Colonial Mutual Buildings, West Street, Durban, in Consulting Dermatological practice, and as from May 1958 their address will be 14th Floor, Provident Assurance House, corner of Smith Street and Field Street. Telephones: 22636 and 61701.

* * *

Dr. I. Freed, of Johannesburg, has been accepted as a postgraduate student to attend the Course in Internal Medicine given by Harvard Medical School for the months of June and July 1958 at the Peter Bent Brigham and the Massachusetts General Hospital. Dr. Freed, accompanied by Mrs. Freed, will leave for Boston by air on 24 May and return on 30 July 1958.

* * *

The 'Jooste Cup' Golf Competition, organized by the East Rand Branch of the Medical Association of South Africa, and open to all members of the Association, is being held this year at Springs Country Club, Springs, on Sunday 13 April. Entries may be submitted to the Convener, Dr. Wm. Sacks, at P.O. Box 813, Springs, or by telephoning 565371 (rooms) or 561138 (residence).

* * *

The Johannesburg Medical Women's Group (a sub-group of the South African Society of Medical Women) is holding a cocktail party in honour of Dr. Alice Cox, on whom an honorary Degree of Doctor of Laws is being conferred by the University of the Witwatersrand, at the residence of the Chairman, Dr. Mary Tomlinson, on Sunday 30 March at 5.30 p.m. All medical women are welcome, including those visiting Johannesburg; and are asked to inform the Hon. Secretary, Dr. E. Neros, of their intention to attend by telephone (22-4571) or post (P.O. Box 2616).

* * *

Glaxo Laboratories, P.O. Box 21, Wadeville, Transvaal, have recently imported three interesting and informative films on research and development of medical interest by their parent company in Britain, and would be happy to lend them free of charge to any organization who may wish to show them to their members. The films are 16 mm., in colour, with sound tract, as follows:

1. 'Thought for Food'—596 feet—running time 15 minutes. This film deals with the planning of a complete food by a nutritional research team—the food in question being Complan which has just been introduced in South Africa.

2. 'A Vitamin Emerges'—560 feet—running time 15 minutes. How Vitamin B12 was isolated at Glaxo Laboratories and how research is increasing our knowledge of this vitamin.

3. 'Longer Life for BCG'—555 feet—running time 15 minutes. The contribution of research to the manufacture and standardization of freeze-dried BCG vaccine.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van gedugte epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 28 Februarie tot 6 Maart 1958.

Pes, Pokkies: Geen.

Tifuskoors. Kaapprovinsie: Twee (2) Naturellegevalle in die munisipale gebied van Steynsburg. Alle voorsorgsmaatreëls getref.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	5	1	–	–	6
Kaapprovinsie ..	2	5	2	–	9
Oranje-Vrystaat ..	–	1	–	–	1
Natal	1	–	–	–	1
Totaal ..	8	7	2	–	17

Local Authorities		*Eur.*	*Non-Eur.*
Transvaal:			
Belfast District	R	1	–
Groot Marico District	R	1	–
Krugersdorp Municipality ..	U	1	–
Piet Retief Municipality	U	1	–
Pretoria Municipality	U	–	1
Vereeniging Municipality	U	1	–
Cape Province:			
Cape Divisional Council	R	–	1
Cape Town Municipality ..	U	1	–
Namaqualand Divisional Council	R	–	1
Queenstown Municipality ..	U	–	2
Riviersonderend Municipality ..	U	1	–
Steytlerville Divisional Council ..	R	–	1
Uitenhage Divisional Council ..	R	–	1
Uitenhage Municipality	U	–	1
Orange Free State:			
Senekal Municipality	U	–	1
Natal:			
Durban Borough	U	1	–

U = Urban. R = Rural.

REVIEWS OF BOOKS : BOEKRESENSIES

HORMONES AND VAGINAL CYTOLOGY

Acquisitions Récentes en Cytologie Vaginale Hormonale. Par J. Paul Pundel, avec la collaboration de Fernand Schwachtgen et Eugène Ost. 236 pages, avec 62 figs. et xv tableaux. 2,400 fr. Paris: Masson et Cie. 1957.

Table des Matieres: Première Partie. La Technique Cytologique des Frottis Vaginaux. *Deuxième Partie.* La Cytochimie de L'Épithélium Vaginal. *Troisième Partie.* Cytologie Vaginale Générale. *Quatrième Partie.* Les Variations Physiologiques de la Cytologie Vaginale de la Naissance a la Postménopause. *Cinquième Partie.* Action des Diverses Hormones sur la Cytologie Vaginale. *Sixième Partie.* Actions de Substances Non Hormonales sur la Cytologie Vaginale. *Septième Partie.* Modifications Cytologiques Bactériennes et Parasitaires. *Huitième Partie.* L'Evaluation Hormonale du Frottis Vaginal. *Neuvième Partie.* Applications Pratiques du Cytodiagnostic Hormonal. Conclusions Générales. Index.

It is only in the last few years that vaginal cytology has attracted much attention, but in a comparatively short time a voluminous literature has accumulated on the subject.

Taking a vaginal smear is a simple, painless procedure and may be repeated as often as necessary. In gynaecology it has great application in the detection and management of endocrine disorders, genital cancer and vaginal infections and infestations.

J. Paul Pundel in his latest publication deals mainly with the endocrinal applications of cytology, his approach being a very practical one. He discusses the advantages of different types of fixatives and staining and the importance of standard methods to produce accurate staining of cells. Normal cell morphology is described in considerable detail and a plea is made for some sort of uniformity in the description of the different cell types. The influence of hormonal factors upon cytology is explained with reference to normal physiological variations and the modifications which may occur due to cytochemical change resulting from vaginal infections are discussed.

This is an extremely practical and informative book written by an expert on the subject and should be read by anyone interested in cytology, be he gynaecologist, endocrinologist or cytologist.

D.M.

PREVENTIVE MEDICINE AND PUBLIC HEALTH

Preventive Medicine and Public Health. Third Edition. By Fred Grundy, M.D., M.R.C.P., D.P.H. Pp. vii + 309. Figs. 37. £1 5s. net. London: H. K. Lewis & Co., Ltd. 1957.

Contents: I. The Scope of Preventive Medicine and Public Health. *Section I.* Administration and Practice. II. Local and Central Government. III. The National Health Service Act, 1846, and Organised Medicine. IV. Social Security and National Insurance. V. The Health Department of Local Authorities. VI. The Medical and Welfare Services of Local Authorities. VII. The Maternity Services and the Midwife. *Section II.* Principles. VIII. The Assessment of Individual and Community Health. IX. Heredity and Health. X. Environment and Health. XI. The Personal Element: Health Education: The Problem Family. *Section III.* Statistics and the Control of Infectious Diseases. XII. Statistical Methods and their Uses. XIII. Certain Statistics of Mortality and Morbidity. XIV. The Mortality of Mothers and Infants. XV. Population and Health Services. XVI. The Control of Infectious Diseases. *Section IV.* Historical and Certain Other Subjects. XVII. Public Health and Social Welfare: Historical. XVIII. International Health Organisations. *Appendix I.* Illegitimacy and Adoption. *Appendix II.* Child Life Protection. *Appendix III.* Nurseries and Child Minders Regulation Act, 1948. *Appendix IV.* Registration and Supervision of Nursing Homes. *Appendix V.* The Rules of the Central Midwives Board. *Appendix VI.* Mental Deficiency and the Intelligence Quotient. *Appendix VII.* Suggestions for Further Reading. Index.

This most interesting, beautifully printed and readable book reveals the fact that its author, a practising health officer, has never permitted this aspect of the whole problem to be lost sight of. He has with great skill drawn to a very great extent on this experience to produce an instructive and informative introduction to preventative medicine and public health. The chapter on epidemiology is wide in scope and very much in keeping with modern thoughts on this most important discipline.

The present health set-up in the United Kingdom receives more than passing reference: its growth and development from the first Health Acts, the direct results of agitation and propaganda by such public health giants as Chadwick, Farr and Simon, are skilfully and most interestingly traced. The mere fact that this little publication has seen no less than 3 editions within the space of 6 years is indeed proof of its general excellence.

The inclusion of an appendix in which are set out suggestions regarding additional reading has much to commend it in a publication of this nature. I have no hesitation in strongly recommending this little book to all interested in public health in its broader aspects.

E.D.

HAEMOLYTIC DISEASE OF THE NEWBORN

Comparative Aspects of Haemolytic Disease of the Newborn. By G. Fulton Roberts, M.A., M.D. Pp. xi + 199. 17s. 6d. net. London: William Heinemann—Medical Books Ltd. 1957.

HAEMOLYTIC DISEASE OF THE NEWBORN——

Contents: Preface. Introduction. I. The Disease in Man: Historical Aspects. II. The Disease in Man: Haemolytic Anaemia. III. The Disease in Man: Kernicterus. IV. Serological Considerations. V. The Disease in the Horse and the Mule. VI. The Disease in the Pig. VII. The Experimental Production of Haemolytic Disease of the Newborn. VIII. Comparative Aspects of Haemolytic Disease of the Newborn. IX. Treatment. A Note on Nomenclature. Appendix. References. Index.

This is an interesting little book. The author is well qualified to write on this topic and has previously written an eminently readable book on the Rhesus factor. In this book he sets out to compare haemolytic disease as it is seen in humans, in the horse, the mule, in the pig and in other animals.

The author disclaims any credit for personal research, the only unaided work described being that on the historical aspects of the disease. Dr. Fulton Roberts has obviously delved deeply into the relevant literature, much of it written in languages other than English, and this alone makes the book very well worthwhile. The description of the disease as seen in man adds little to what is generally taught, but medical men will be grateful for the sections on the disease in animals. They will once again see just how similar disease in animals can be when compared with that occurring in humans. We are becoming accustomed to seeing advances in medicine being applied to animal work and would wish that there could be more 'traffic in the reverse direction'. For there must be many unrecognised principles of disease in animals which have a bearing on the disease in man. It is books like this one which bring out how much each of these sciences stands to gain from the other and one would wish there were more books like it.

C.M.

POSITIONING IN RADIOGRAPHY

Positioning in Radiography. Seventh Edition. By K. C. Clark, M.B.E., F.S.R. Pp. 655 with 2,150 illustrations. 105s. London: William Heinemann (Medical Books) Ltd. 1956.

Contents: Preliminary Note. 1. Upper Limb. 2. Humerus and Shoulder Girdle. 3. Lower Limb. 4. Hip Joint and Upper Third of Femur. 5. Pelvic Girdle. 6. Vertebral Column. 7. Vertebral Column. 8. Bone of Thorax. 9. Skull. 10. Mandible. 11. Salivary Glands. 12. Air Sinuses of the Skull. 13. Optic Foramina. 14. Lacrimal Ducts. 15. Temporal Bones. 16. Ventriculography and Encephalography. 17. Subject Types. 18. Heart and Aorta. 19. Kymography. 20. Respiratory System. Thymus Gland. 21. Tomography. 22. Alimentary Tract. 23. Abdomen. 24. Gall Bladder. 25. Urinary Tract. Prostate. 26. Female Genital Organs. 27. Foreign Bodies. 28. Dental. 29. Soft Tissue. 30. Myelography. 31. Stereography. 32. Miniature Radiography. 33. Cineradiography. 34. Macroradiography. 35. Angiography. *Supplements.* 1. Contrast Media. 2. Note on the Exposure Tables. 3. Radiation Exposure to the Patient. 4. Metric Equivalents.

In her seventh edition of 'Positioning in Radiography', Miss K. C. Clark has continued to display her superb ability to produce a really magnificent text book of radiographic techniques.

Each previous edition has been regarded as a standard reference for the radio-diagnostic department, and this reputation is further enhanced by the present publication. Every position which can be utilised for radiographic examination of all sections and systems of the body is fully described and the necessary exposure factors are given. Some 600 new illustrations have been added and, as before, the reproductions leave nothing to be desired.

The text has been increased by 120 pages.

Details of macro-radiography, although not widely employed, are a useful addition.

Cineradiography has been described more fully and image intensification is discussed.

A chapter on radiation exposure to the patient is of particular importance, in view of the need for the fullest appreciation of radiation hazards.

It is thought that a description of protective devices for the patient would have been an advantage, but no doubt this omission will be rectified in the next edition.

The highest praise must be given to Miss Clark for her most valuable publication. The book is an essential for the training and guidance of radiographers. To all who do diagnostic radiology, it is a mine of information.

J.J.N.

HUMAN FERTILITY AND STERILITY

Studies on Fertility 1956. Volume VIII. Edited by R. G. Harrison, M.A., D.M. Pp. x + 138. Illustrations. 25s. Oxford: Blackwell Scientific Publications. 1957.

Contents: The Male. (*a*) Human Fertility. I. The Use of Partitioned Ejaculates in Investigating the Role of Accessory Secretions in Human Semen, *by* Clare Harvey. II. Pregnancy following Artificial Insemination from Epididymal Cyst, *by* H. G. Hanley. III. Testicular Biopsy in Sterile and Subfertile Males, *by* J. H. Carver. (*b*) Fertility in the Experimental Animal. IV. The Effects of Unilateral Orchidectomy on the Rat Testis, *by* J. H. Grant. V. Studies relating to the Storage of Mammalian Spermatozoa, *by* I. G. White. VI. The Influence of Seminal Fluid on Fertility, *by* N. T. Werthessen, W. Marden, F. Haag and J. W. Goldzieher. VII. The Initiation of Motility in Mammalian Spermatozoa, *by* A. Walton. VIII. The Mechanism of Spermiation in the Male Frog, *Rana esculenta*, *by* J. Botella-Llusia, F. Plaza and J. R. Del Sol. IX. The Immediate Effects of Occlusion of Superior Epididymal Arteries in the Rat as Demonstrated by the use of Radiopaque Medium, *by* E. W. Macmillan. X. Thermoregulation of the Testis at High Temperature, *by* G. A. Harrison and R. Harris. *The Female.* (*a*) Human Fertility. XI. The Use of a New Long-acting Progestational Compound (17-Alpha-Hydroxyprogesterone-Capronate) in Infertility and Habitual Abortion, *by* E. T. Tyler. XII. Brief Evaluation of some Therapeutic Measures used at an Infertility Clinic during the past Twenty Years, *by* M. C. N. Jackson. (*b*) Fertility in the Experimental animal. XIII. An attempt to produce the Hertwig effect by X-irradiation of male mice, *by* H. M. Bruce and C. R. Austin. Subject Index. Author Index.

Most of the papers in this volume were read at a meeting of the Society for the Study of Fertility held in London in 1956. Results from both the clinical and the experimental fields of investigation are presented. Of the thirteen chapters in the book ten are devoted to studies of fertility in the male.

The section on the male includes an interesting report by H. G. Hanley of St. Paul's Hospital, London. He finds that in cases in which azoospermia is due to congenital abnormalities, e.g. absence of the vasa, an artificial spermatocele can be formed by the insertion of a loosely folded ball of amnion inside the head of

the epididymis. From such an artificial epididymal cyst, spermatozoa capable of fertilising the ovum can be aspirated.

Studies by E. T. Tyler of the School of Medicine, University of California, indicate that a new steroid ester, 17-alpha-hydroxyprogesterone caproate, is of value in the treatment of infertility due to 'luteal defects' and in habitual abortion. This new steroid is a highly potent long-acting progestational compound which can be administered in large doses with relatively little side effects.

The major part of this symposium is devoted to fertility studies in the experimental animal and as such may have a less immediate appeal to the clinician. On the whole, however, both experimentalist and clinician will find the results recorded in this volume of great interest.

H.J

BAIRD'S TEXTBOOK OF OBSTETRICS AND GYNAECOLOGY

Combined Textbook Obstetrics and Gynaecology. Sixth Edition. Edited by Dugald Baird, B.Sc., M.D., D.P.H., F.R.C.O.G. Pp. xii + 936. Figs. 492 (27 in colour). 95s. net + 2s. 6d. postage abroad. Edinburgh & London: E. & S. Livingstone Ltd. 1957.

Contents: Part I. Anatomy of the Genital Organs. I. Anatomy of the Female Pelvis and the External Organs of Generation. II. Anatomy of the Internal Genital Organs—Female. *Part II. Physiology of the Reproductive System.* III. Physiology of the Reproductive System. IV. Development of the Embryo and Membranes. V. Anatomical Changes resulting from Pregnancy. VI. Physiology and Nutrition in pregnancy and lactation. VII. The Factors of Labour. VIII. The Phenomena of Labour. IX. Normal Puerperium. X. The Newborn Infant. *Part III. Management of Pregnancy and Labour.* XI. Examination of the Reproductive System: General Procedure in Gynaecology and Obstetrics. XII. Signs and Symptoms—Differential Diagnosis of Pregnancy. XIII. The Duration of Pregnancy—Causes of the Onset of Labour. XIV. Antenatal Care. XV. Preparation for and Management of Labour. XVI. Sedatives—Analgesics—Anaesthetics. XVII. Management of the Puerperium and Post-Natal Care. XVIII. The Care of the Newborn. *Part IV. Pathology of Pregnancy.* XIX. Abortion or Miscarriage. XX. Abnormalities of Implantation of the Ovum—Pregnancy in Tube.—Peritoneum—Pregnancy in Rudimentary Horn—Angular Pregnancy. XXI. Diseases and Abnormalities of the Ovum. XXII. The Association of Pregnancy with Systemic Disorders. XXIII. Disturbances of the Reproductive System Complicating Pregnancy. XXIV. Vomiting in Pregnancy. XXV. The Toxaemia of Pregnancy. XXVI. Haemorrhage in Later Months of Pregnancy. XXVII. Dystocia. XXVIII. Dystocia due to Faults in the Child. XXIX. Complications arising from Abnormalities of the Umbilical Cord and Membranes. XXX. Injuries to the Birth Canal and Other Accidents to the Mother. XXXI. Third Stage and Postpartum Dystocia. XXXII. Post-Haemorrhagic Collapse and Shock in Obstetrics: Blood Transfusion. XXXIII. Complications of the Puerperium. XXXIV. Birth Injury and Anoxia. XXXV. The Premature Infant. XXXVI. The Physiological Adjustments of Neonatal Life and their Dangers. XXXVII. Malformations in the Newborn. XXXVIII. Infections in the Newborn. XXXIX. Obstetric Operations. XL. Vital Statistics and Reproduction. *Part V. Gynaecology.* XLI. The Investigation of the Gynaecological Patient. XLII. Disorders of Female Sex Function. XLIII. Malformations of the Reproductive Organs. XLIV. Displacements of the Uterus. XLV. Infections of the Reproductive Organs. XLVI. Tumours of the Vulva, Urethra and Vagina. XLVII. Diseases of the Uterus. XLVIII. Diseases of the Ovary. Broad Ligament and Fallopian Tubes, excluding Inflammatory Disease. XLIX. Endometriosis. L. Sterility. LI. Family Planning. LII. Psychological Aspects of Midwifery and Gynaecology. LIII. Some Medico-Legal Considerations. *Part VI. Gynaecological Operations.* Index.

The publishers have given admirable execution to the wish expressed in the Editor's preface, that this new edition should be more convenient to handle than its rather cumbersome predecessor. By closer print and spacing, as well as an increase in width of page, the new volume has been reduced by a full inch in thickness and is almost 400 pages smaller and in thus following the modern trend in medical textbook publications, permits easy handling even by women students and practitioners. Also following recent trends, there is less anatomy, more physiology; fewer drawings but more and better photographs.

All this has been accomplished without loss of essential text, much of which has been rewritten, incorporating recent advances. Amongst these, the pioneer work done in Aberdeen on Obstetric and Gynaecological epidemiology is in welcome evidence, notably in the chapters on dystocia, vital statistics, and carcinoma of the cervix. 'However great may be our aversion to figures we cannot escape the conclusion that the solution of most of the problems of clinical or preventive medicine must ultimately depend on them.' On the other hand, the importance of the psychological aspect of the patient, and of the personal relationship between her and her doctor is stressed even more than before. The contribution of the National Health scheme in making the best hospital care plus a great deal of personal attention available even to the poorest of patients, is pointed out in the chapter on antenatal care, one well worth re-reading at regular intervals by anyone engaged in obstetric practice, be he or she student, general practitioner or consultant.

This book breathes the very spirit of Sctottish Obstetrics and Gynaecology—cold logic on the one hand, the humane touch on the other. It is this spirit which has made possible over the past half-century the immense Scottish contributions to what we now know as antenatal care.

J.P.R.

CORRESPONDENCE : BRIEWERUBRIEK

A SUNBURN OINTMENT

To the Editor: Recently given a tube of 'Sea and Ski' a 'tanning cream' made by Rolley, Inc., of San Francisco, by a sympathetic chemist who noticed my raw and desquamating face, for the first time in years of fruitless and costly experimentation with every alleged protective agent from pills to pastes, I avoid the standard penalty of one epidermal layer and 3 days' torture per hour's exposure to sun. Rapidly absorbed, with a transient, pleasant smell, one application sees me through a sunny day on the river and is 100% protective. Noted as containing glyceryl p-amino benzoate, this may mean that I slept through an organic chemistry and a dermatology lecture—both very likely, but plenty of colleagues must have been equally somnolent and I feel sure some of your readers might like to give this preparation a trial. It entirely amazed me.

Sunstroke

11 March 1958

ATHERSTONIAN SOCIETY

To the Editor: The appearance in the *Journal* of Dr. Schmidt's essay on the History of Anaesthesia in South Africa and in the same issue the publication of Dr. Glauber's paper 'A Scheme for the Valuation of Anaesthetics', which was originally read before the Atherstonian Society, prompts me to explain the origin and purpose of this Society.

Originally the registrars of the Departments of Anaesthesia at the King Edward VIII Hospital and Addington Hospital, Durban, met at the home of one of our members (Dr. Grant-Whyte) for clinical discussion amongst themselves. In time anaesthetists from other hospitals and in private practice were invited to these informal meetings. Eventually it was decided to form a Clinical Society for all practitioners in Natal interested in the science and art of Anaesthesia. I had the good fortune to act as convener and chairman of the first meeting.

In commemoration of what we considered to be the first notable scientific achievement in the practice of Anaesthesia in South Africa, viz., the contribution by Dr. W. Guybon Atherstone at Grahamstown on 19 June 1848, it was decided to name the Society the Atherstonian Society.

The aim of the Society is to foster the science and art of Anaesthesia, to enable house officers and registrars to discuss their problems and difficulties with senior colleagues, to encourage younger members to speak at clinical meetings, and to bring full-time hospital anaesthetists, specialists in private practice, and general practitioners interested in the practice of Anaesthesia into closer clinical contact.

I am happy to say that the Society, now entering its second year, is flourishing.

Hugh Foord

515 Sanlam Building
Smith Street
Durban

6 March 1958

Glauber, D. (1958): S. Afr. Med. J., 32, 236 (1 March).
Schmidt, H. J. (1958): *Idem.*, 32, 244 (1 March).